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ANATOMY OF THE EIGHTH NERVE.

The Central Projection of the Nerve Endings of the Internal Ear.*

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Introduction.

The anatomical studies, the publication of which begins with this paper, have been accomplished in different laboratories: Instituto Cajal (Madrid), Otological Clinic (Upsala), Kaiser Wilhelms Institut für Hirnforschung (Berlin), Casa de Salud Valdecilla (Santander) and Central Institute for the Deaf (St. Louis, Mo.). Although they were started in 1922 and never interrupted since, only a few of the established facts have been published. The nature of the problems justifies the time and material used.

I.—THE PROJECTION OF THE COCHLEA IN THE PRIMARY ACOUSTIC NUCLEI.

1. The Problem.

In establishing the theories of hearing, special attention has been paid to the physics of the cochlea, but only very little to the central mechanism.

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It is evident that the cochlea in its function has to obey the laws of Physics and, therefore, it is perfectly justified to consider it as a physical apparatus in order to determine how it responds to the sound vibrations. But what happens in the cochlea is not hearing: in the cochlea, as a result of its activity, only nerve impulses are produced which must travel by the long hearing tract and traverse at least three stations (primary acoustic nuclei, internal geniculate body and primary acoustic cortex) before the sensation of hearing arises. How the cochlea vibrates is independent of the nervous system, but how its vibrations are recorded in the brain depends entirely upon the anatomical and physiological properties of the hearing pathways. We consider, for instance, the Helmholtz theory. With this theory or any of those that are based on the assumption of resonance of a limited part of the cochlea with each tone, a subsidiary assumption must be made, namely, that each point of the cochlea has individual fibres all along the hearing tract. Whether this is true or not is still unknown.

If the telephone theories or M. F. Meyer theory are considered, the necessity of a perfect knowledge of the hearing tract does not need to be emphasized, for according to these theories the analysis of sounds should be chiefly a central phenomenon.

The general features of the hearing pathways are well known, and the reader may find excellent information about them in the books of Cajal,[10] Herrick,[23] Kappers[26] or Ranson.[41] But as to the fine details, even as to many fundamental questions, it can be said that the problems are still totally open.

The first problem that offers itself to our consideration is whether in the hearing pathways individual fibres for the different regions of the cochlea are present.

2. Historical.

The great Swedish neurologist, Henschen,[22] after having established that the retinal macula is projected in a relatively small region of the visual cortex, postulated on the basis of several cases of brain lesions the existence of a projection of the organ of Corti in the primary acoustic cortex. However, the available evidence was undoubtedly not sufficient and this question has been matter for repeated discussions about which the reader may find information in the paper of Börnstein.[5] It seems that the study of clinical material is not sufficient to solve the problem. Recently, experimental work has furnished evidence in favor of the view that the corpus geniculatum internum is projected in the primary acoustic cortex. Pfeiffer,[34] working with the Flechsig myelinisation method, found

that the acoustic radiation shows a very regular arrangement of its fibres that makes an anatomical projection of the geniculate body very likely. Direct proof, however, was not available until the recent work of Poljak[39, 40] who, making use of the Marchi's methods, has found, with the monkey, that after circumscribed lesions of the primary auditory region, only small, circumscribed regions of the internal geniculate body degenerate. The complete publication of Poljak's experiments must be awaited with utmost interest.

But that the internal geniculate body be projected in the primary acoustic cortex does not necessarily implicate that a similar projection exists in the rest of the acoustic pathways. There are still two links in the chain that have to be examined, the projection of the geniculate body in the primary acoustic nuclei and that of the cochlea in these nuclei. A study of the projection of the primary acoustic nuclei in the internal geniculate body has never been accomplished.

On the projection of the cochlea in the primary acoustic nuclei there are data, furnished by Poljak[35, 38] and myself.[30, p. 39; 31] Poljak, using the Weigert's method with the bat, found a very regular arrangement of the cochlear fibres and postulated the existence of an anatomical projection of the cochlea: the fibres from the apex should end in the ventral region and those from the basis in the dorsal one of the ganglion ventrale. The results of my own studies were occasionally mentioned in 1925 and 1927, but I waited for a complete publication, because I thought that the more extensive the study, the greater would be the guaranty of having found the solution of the problem. As a matter of fact, it was in 1924* that I found proof of the projection of the ganglion of Corti.

3. Materials and Technique.

Theoretically, the ideal method for determining the anatomical projection of the cochlea should be Marchi's method. Practically, however, this method is useless because, although it is possible to make extirpations of only a part of the cochlea, the degenerations do not remain limited to the fibres from the destroyed part. Therefore, there is no other possibility than to use the anatomical methods in suitable material.

The methods used have been those of Golgi, Cajal and Weigert-Kulschitzky in the customary way.

The most suitable material are mice because it is possible to prepare serial sections, stained with one of the mentioned methods,

*Communication to the Congress of Scandinavian Neurologists in Stockholm, September, 1924.

through the internal ear and the medulla oblongata, in which the course of the cochlear fibres may be followed either in a single or in a few sections. Embryos of the mouse or cat stained after Cajal also give excellent preparations.

The anatomical projection of the cochlea was studied first in the mouse with the help of Golgi's method. It happens quite often that from the whole cochlear nerve only a small group of fibres are stained, and in such cases it is extremely easy to follow those fibres from the ganglion of Corti to their termination in the centres.

In serial sections from the mouse stained after Weigert-Kulschitzky, or after Cajal, the whole cochlear nerve may be followed, but, of course, there are no longer the individual fibres but the bundles of fibres. Reconstruction in glass plates of the cochlear nerve and its division branches in the primary nuclei may be easily made; in such reconstructions the twisted course of the cochlear nerve appears in plastic form.

With the cat I do not have direct proof of the anatomical projection of the cochlea because I do not have series of sections through the medulla and internal ear. But since the course of the cochlear nerve within the temporal bone and within the medulla have been found to be extremely similar as in the mouse, I have no doubt that the same projection of the cochlea is present. On the other hand, since the cochlea develops in the cat in the same way as in the mouse, the course of the cochlear nerve and the projection of the ganglion of Corti must necessarily be the same.

4. The Central Ending of the Cochlear Fibres.

The cochlear nerve enters the medulla lateral to the descending root of the trigeminus; its fibres immediately divide into ascending branches that go to the ganglion ventrale and descending ones that go to the tuberculum acusticum. This division was discovered by Held;[19] Kölliker[27] confirmed its existence and Cajal[7, 10] gave a very complete description. However, the drawings of these authors do not show a very remarkable arrangement which is a consequence of the anatomical projection of the ganglion of Corti.

All the ascending and descending branches are arranged in parallel bundles, and if the points of division of the root fibres are projected on a longitudinal plane, they form an arc line of anterior concavity. That is the caudal boundary of the ganglion ventrale. This arrangement can be seen in Fig. 2 that is a diagram, and in Fig. 1 that reproduces the cochlear fibres stained in a longitudinal section of the acoustic ganglia.

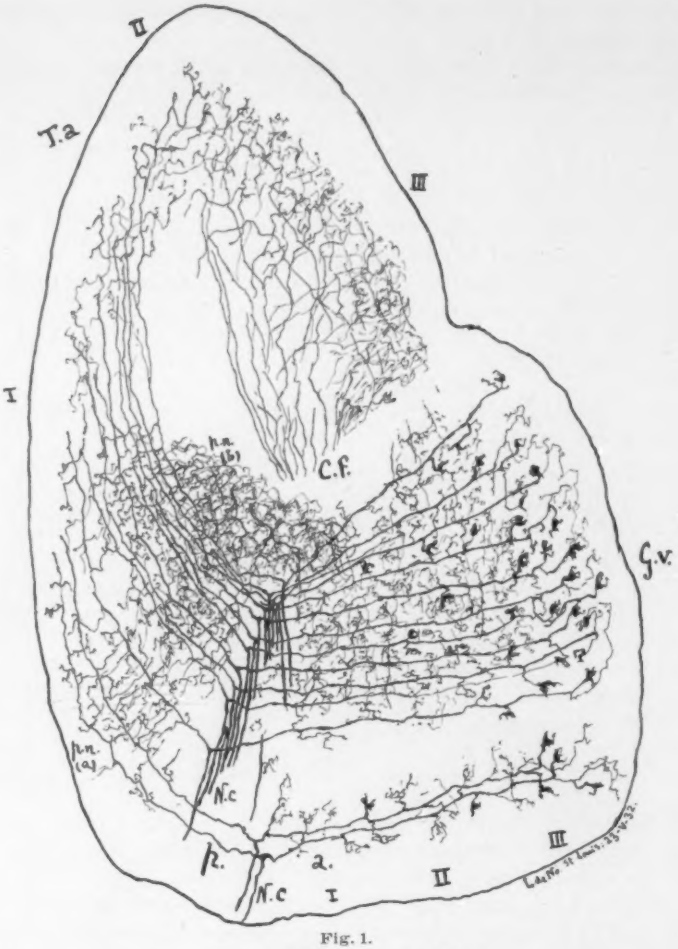

Fig. 1.
See legends at end of article.

5. The Course of the Cochlear Nerve from the Ganglion of Corti Into the Medulla.

The cells of the ganglion of Corti are distributed all along the cochlea, thus constituting a spiral that runs parallel to the organ of Corti itself. From the outer edge of the spiral ganglion arise the peripheral processes of the cells which end in the organ of Corti;

from the inner edge arise the central processes which form the cochlear nerve.

It is now very important that this nerve is not arranged as any other in parallel bundles of fibres but is twisted on its own axis in the same direction as the cochlea itself, so that each fibre shows with reference to the axis as many turns as that part of the ganglion of Corti, from which it arises, shows with reference to the base of the cochlea, or better said to that part of the cochlea that first appears in the embryo. In other words, if the nerve is followed from the point of division of its fibres in the medulla, towards the ganglion of Corti, it is found that it twists progressively as it advances, and each bundle of fibres turns on the axis of the nerve in the same direction

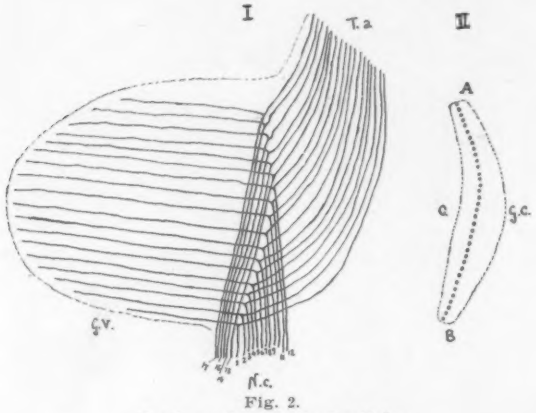

Fig. 2.
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of the cochlea, the number of turns being the same of that part of the ganglion of Corti where it enters. This is why the arc line formed by the points of divisions of the fibres in the medulla really is the projection of an uncoiled ganglion of Corti, the highest point corresponding to the apical end of the ganglion and the lowest one to the basilar part (see Fig. 2).

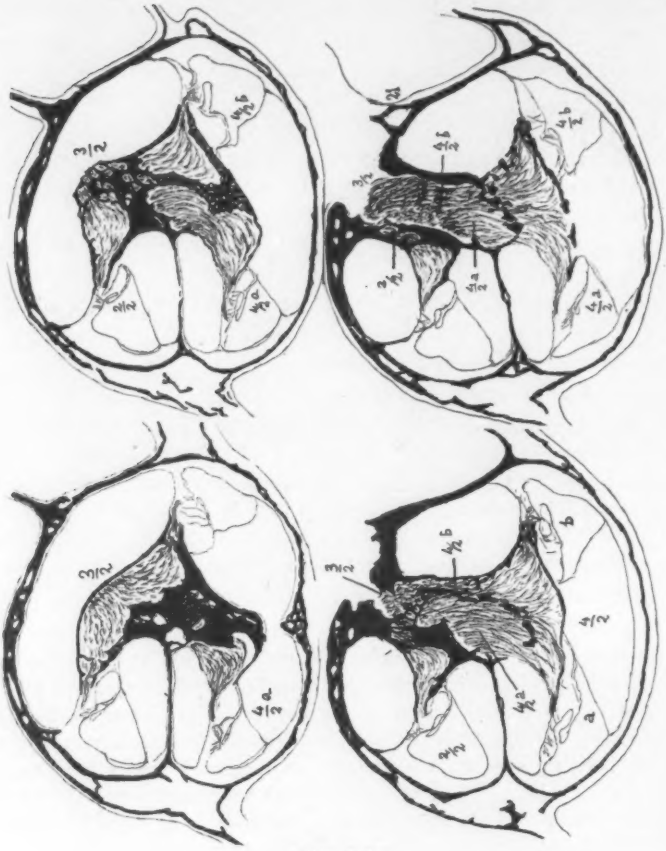
Under the microscope it is not difficult to follow with the help of a serial reproduction in drawings (see Figs. 3 to 12) or in photographs the individual bundles from those points of divisions to the ganglion of Corti; the task is still easier if a glass reconstruction of the nerve is made, because all the parts of the bundles contained in each section may be observed at the same time; but it is very difficult to give a satisfactory reproduction of the course of the nerve.

The drawing in Figs. 3 to 12 give an idea of the general arrangement, but are obviously insufficient for a complete reproduction.

The cochlea of the mouse has very nearly two spirals, therefore in the drawings in Figs. 3-12 the bundles have been marked with $\frac{4}{2}$ (a, b) $\frac{3}{2}, \frac{2}{2}, \frac{1}{2}, \frac{1}{4}$; the turns of the bundles appear in the drawings as changes in their relative position in the successive sections, for instance, the turn of bundle $\frac{4}{2}\,\underset{\sim}{a}$ on bundle $\frac{4}{2}\,\underline{b}$ is clear in Figs. 3 to 8; the turn of bundle $\frac{4}{2}$ on bundle $\frac{3}{2}$ appears in Figs. 3 to 7, the twisted course of bundle $\frac{1}{4}$ is to be seen in drawings 12 to 10; the bundle $\frac{1}{2}$ shows almost no twisting.

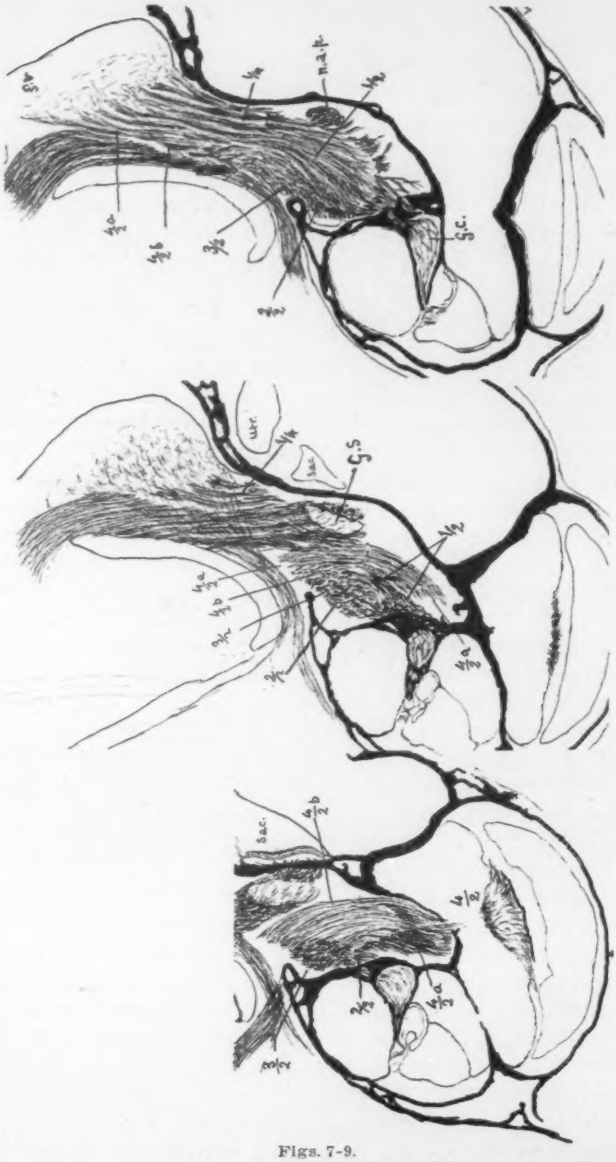
A further analysis of this projection of the ganglion of Corti in the primary acoustic nuclei is obtained with Golgi stains in which only small bundles of the cochlear nerve are stained. I have been able to study numerous preparations of this kind and therefore to follow the fibres almost from any region of the ganglion into the medulla; the results have always been in agreement with the foregoing description.

Only one fact more shall be mentioned here. With the cat the ganglion ventrale has in longitudinal sections the form indicated in Fig. 2. Therefore, the part of the ganglion in connection with the fibres of the central region of the cochlea is in proportion bigger than that in connection with the apex or the base; therefore, if a fibre from the apex or from the base is connected with, for instance, 50 cells in the ganglion ventrale (to be sure with many more) a fibre from the central part of the cochlea will be in connection with 75 or 100 cells. This is in agreement with the results obtained by Guild and coworkers[17, 18] in their studies on the number of cells of the ganglion of Corti; they have found that in the central region of the cochlea there are more ganglion cells per unit length of the organ of Corti than in the basal or apical regions. Thus, the innervation of the central region of the organ of Corti in which most likely the central part of the tone scale is perceived is richer; now we find that in the primary acoustic nuclei the fibres from the central region of the ganglion of Corti again have at their disposal more nerve cells for the transmission of the impulses they carry. The physiological signification of these facts undoubtedly is that the cen-



Figs. 3-6.
See legends at end of article.

tral region of the cochlea will be able to discriminate more acutely between stimuli (between smaller differences in pitch or intensity) than the basal or apical parts. With the mouse and rabbit the different sections of the ventrale ganglion have approximately the same width, *i. e.*, contain the same number of nerve cells; therefore, it is to be expected that the sensitivity of the cochlea be approximately the same in all its regions.



Figs. 7-9.
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6. An Embryological Explanation of the Course of the Cochlearis Nerve.

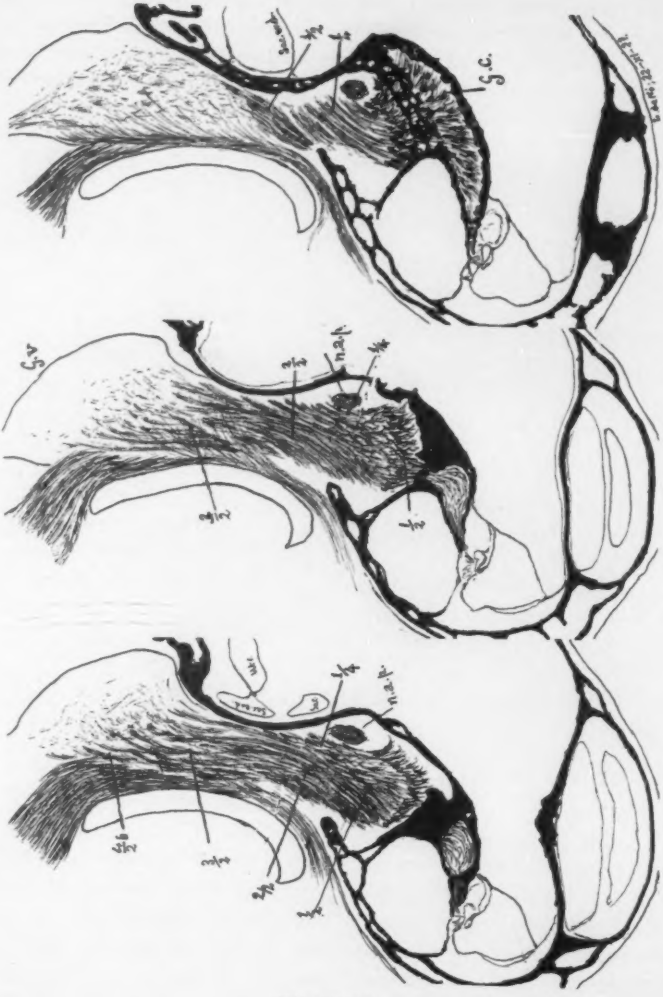
It is not intended to give here a detailed description of the development of the cochlea and the ganglion of Corti, but only to mention some facts that may help in understanding the twisted course and regular arrangement of the cochlear fibres. Classical monographs with complete information about the embryology of the cochlea and the ganglion of Corti are those of Böttcher,[6] and Streeter;[42] additions to the bibliography on the development of the nerve endings may be found also in the papers of Cajal[11] and myself.[30]

The cochlea develops as a process of the vesicula auditiva that gradually lengthens and at the same time coils on itself. But long before the organ of Corti makes its appearance, the ganglionic cells of Corti migrate and place themselves on the medial side of the vesicula auditiva. This stage in which the ganglion of Corti exists without the organ of Corti lasts very long; for instance, with the mouse until the embryo is some 7 to 8 m.m. long.

The central processes of the Corti's cells develop very soon; the moment in which they first appear has not yet been determined, but there is no doubt that they are present in very early stages. With the mouse in embryos of some 5 to 6 m.m. length the central branch of the cochlear nerve is very apparent, and everything indicates that it developed much earlier. But this problem does not concern us here. Now it is important to note only that the central branches of the Corti's ganglionic cells enter the medulla and divide into ascending and descending branches before the organ of Corti appears. Fig. 13 represents a section through the head of a cat embryo in which the organ of Corti is beginning to appear, although no nerve fibres have penetrated it yet, but the cochlear nerve already constitutes a thick bundle, undoubtedly containing all its fibres; it has already penetrated into the medulla and established connections; the division cannot be seen in Fig. 13, but the descending branches and their ramifications in the tuberculum acusticum are very apparent.

Since the cochlear fibres, by means of their ramifications, become anchored in the medulla, their central arrangement will not change and in the following stages of development only the trunk of the nerve will undergo special modifications. In the stage reproduced in Fig. 13, the cochlear fibres still run parallel in the nerve trunk, afterwards they will become twisted.

The ganglion of Corti migrates at the same time that the cochlea develops (S. Boettcher, Streeter). Using a perhaps not quite correct expression, we may say that the ganglion migrates with the apex of



Figs. 10-12.

See legends at end of article.

the cochlea, leaving after it the cells that innervate the regions of the cochlea the ganglion has already passed. Since the cochlea coils itself the nerve trunk becomes twisted and each fibre shows with reference to the axis of the nerve as many turns as that point of the cochlea which it innervates; untwisted will be only those fibres that innervate the region of the cochlea that does not make any translation.

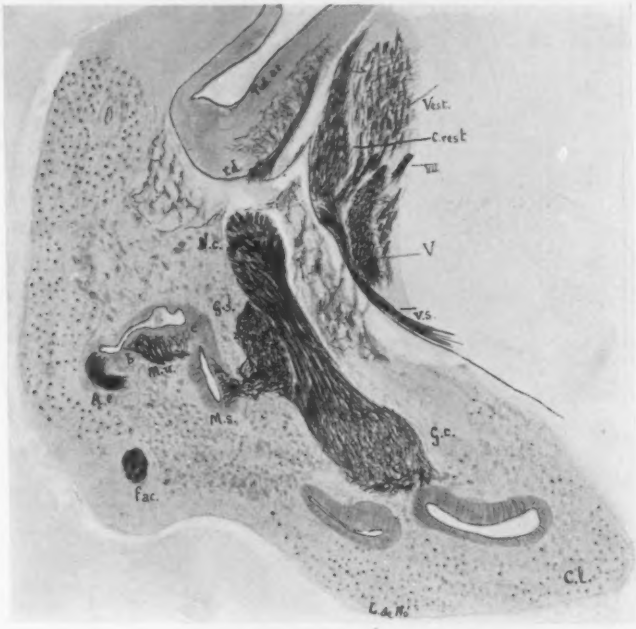


Fig. 13.

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The development of the cochlea takes place not only towards the apex, but also in small proportion towards the base,[30, p. 84] so that the point of the cochlea that does not show any actual displacement corresponds, in the mouse, approximately to the first third of the basilar spiral; the fibres innervating it do not show any twisting and constitute the axis of the cochlear nerve. The fibres innervating the base are twisted in the opposite direction and those innervating the apical part in the same direction as the cochlea.

7. Discussion.

That the ganglion of Corti be projected on the arc line formed by the points of divisions of the cochlear fibres does not signify that each point of the organ of Corti is projected in one and only one point of this line. On the contrary, every point of the organ of Corti is projected in several others.

In the cochlea there are several kinds of endings of which Retzius,[42] Held,[20] v. Gehuchten[14] and Cajal[7a, 10] gave very good descriptions; lately Kolmer[28] Held[21] and Poljak[36] have found them again. Cajal,[11] Tello[44] and I[30] have examined their development.

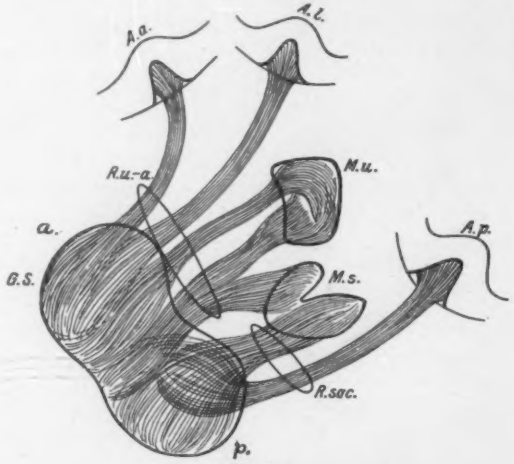

Fig. 14.

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The different kinds of fibres are the following:

1. The radial or internal fibres that go straight to the organ of Corti and end on the internal hair cells; each fibre has connections with only a few neighbor cells and the branches of a few fibres constitute a neuro-fibrilar scale for one hair cell.

2. The spiral external fibres that once arrived at the level of the external hair cells bend in a right angle towards the base of the cochlea and constitute spiral bundles underneath the hair cells; it is very difficult to determine how far each fibre goes within the spiral bundle, but in many Golgi preparations I saw fibres as long as a third of a spiral or even longer. During their spiral course they

establish connections with the hair cells, so that each fibre receives impulses produced in many hair cells distributed in a long segment of the cochlea.

3. The internal spiral fibres constitute a thick bundle of extremely fine fibres underneath the internal hair cells. Their origin is unknown because the silver methods are unable to furnish reliable information and there is no complete description of these fibres based on Golgi preparations. I intend to publish results of my studies on this question in a special paper.

4. There are in the ganglion of Corti spiral bundles that run apicalwards for variable lengths before they bend themselves toward the organ of Corti. The nature of these bundles is unknown.

5. Cajal has described fibres that begin to ramify in the interior of the ganglion of Corti. The endings of these fibres also are unknown.

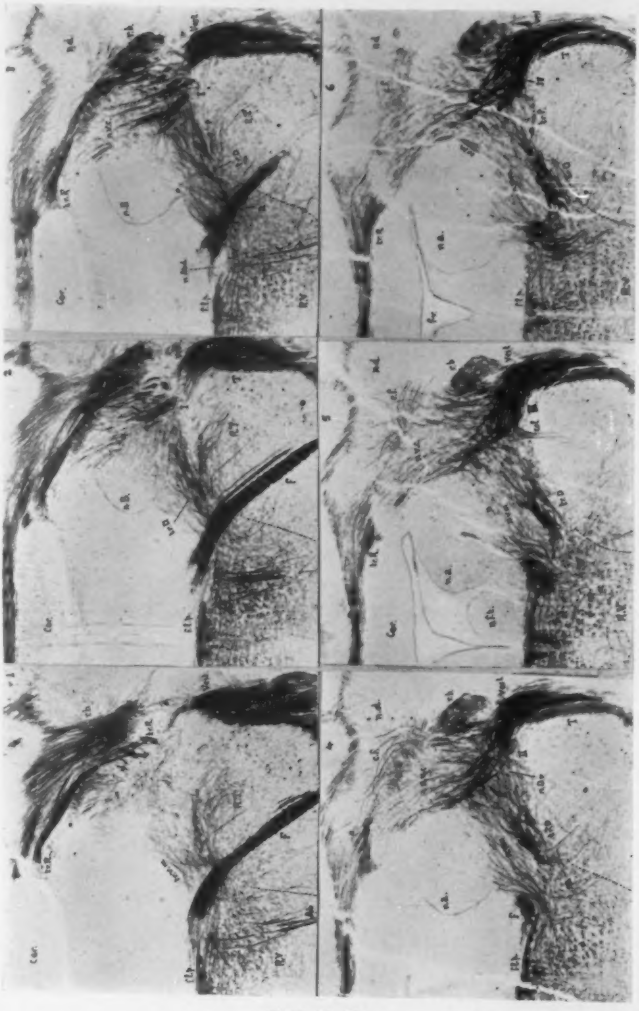
Since so many things still are unknown, it is impossible to determine in detail how the cochlea is projected in the primary acoustic nuclei but on the basis of the available information it can already be stated that the projection is not point-like, because each point of the cochlea is represented several times in the primary acoustic nuclei, once by means of the radial fibres innervating the internal hair cells, and several times by each one of the spiral fibres that cross underneath the external and internal hair cells.

This does not, by any means, signify that the Helmholtz theory or its modifications are impossible; as a matter of fact many subsidiary assumptions can be made that would fit into both theory and anatomical arrangement, but it does mean that the hearing theories must consider the function of the nervous system. Within the Helmholtz theory the necessity of nervous processes correcting imperfections of the peripheral organ has been emphasized among others by Bárány,[2] Fletcher,[12] Poljak,[30] etc. On the other hand, a point of the cochlea, although represented several times in each of the primary acoustic nuclei might, at the same time, be represented by individual fibres in the secondary hearing tract that goes towards the internal geniculate body. Only further researches may throw light on this problem.

Conclusions.

1. An anatomical proof of the projection of the ganglion of Corti in the primary acoustic nuclei is given.

2. The cochlear fibres, shortly after their arrival in the medulla, divide into ascending and descending branches; the points of division of the fibres, if projected on a longitudinal plane, form an arc that



Figs. 15-20.

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is the caudal boundary of the ventral nucleus. This line is a projection of the uncoiled ganglion of Corti in such a way that the highest point of it corresponds to the apical and the lowest to the basilar part of the ganglion.

3. Owing to the existence of different kinds of endings, the cochlea is not projected point-like in the primary acoustic nuclei, because each point of the cochlea is represented several times.

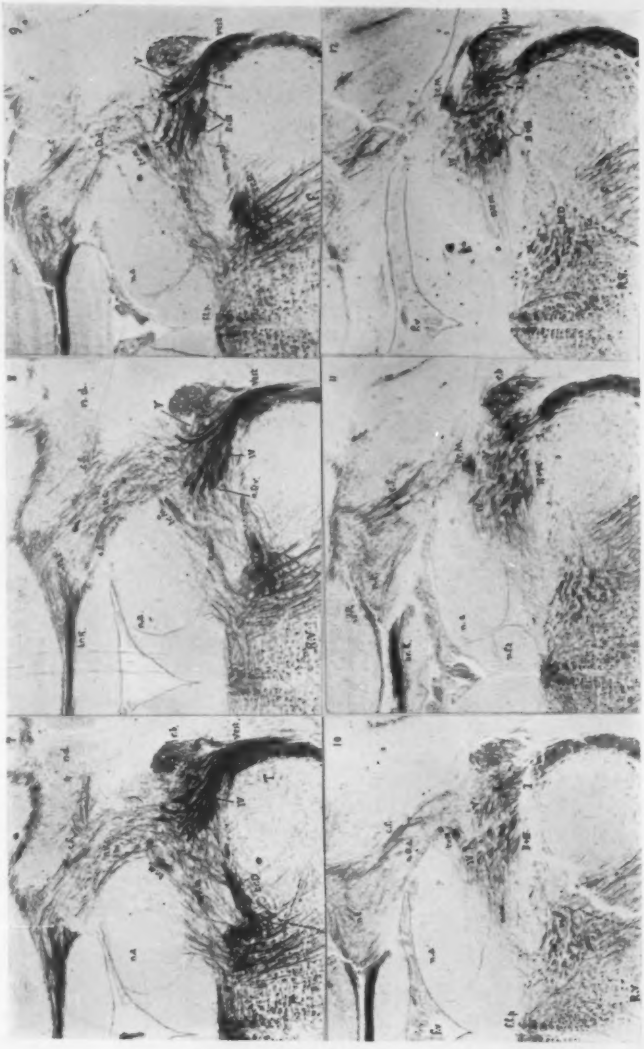
4. An embryological explanation of the twisted course of the cochlear nerve is given.

II.—THE PROJECTION OF THE STATIC LABYRINTH IN THE PRIMARY NUCLEI IN THE FLOOR OF THE FOURTH VENTRICLE.

1. The Problem.

The cells of the ganglion of Scarpa have two processes, a peripheral process going towards the end-organs in the static labyrinth and a central one going into the medulla oblongata; the peripheral process receives the stimuli produced in the labyrinth and the central one carries them toward the centres. In the peripheral division of the vestibular nerve different classes of fibres are known; in the first place, the fibres innervating each one of the terminal end-organs (cristae of the semicircular canals, maculae) constitute a special nerve and, on the other hand, in each organ different kinds of endings have been found. (S. the newest findings and the old literature in the papers of Poljak[37] and myself.[30, 32]) But whether in the central division of the vestibular nerve different classes of fibres are present is not known.

This is a fundamental problem of considerable importance for the understanding of the physiology of the labyrinth. If all the end-organs of the labyrinth were connected with the same nerve cells, they should have very similar functions, the differences being conditioned chiefly by the mechanism of peripheral stimulation; but if each end-organ should have an especial function, the central representation ought to be different. For instance, in recent years the failure to obtain definite symptoms after extirpation of the saccular macula has led several authors to the assumption that this macula has not a static function, but rather plays a rôle in hearing. That no static disturbance is found after the operation is a fact that may have two alternative explanations; either the saccular macula has no static function or our knowledge is so small that we are not able, with our poor methods, to detect the disturbance following its extirpation. To choose between the two alternatives is, as long as no better physiological methods are devised, extremely difficult. But if it should be possible to demonstrate that the fibres arising from the



Figs. 21-26.

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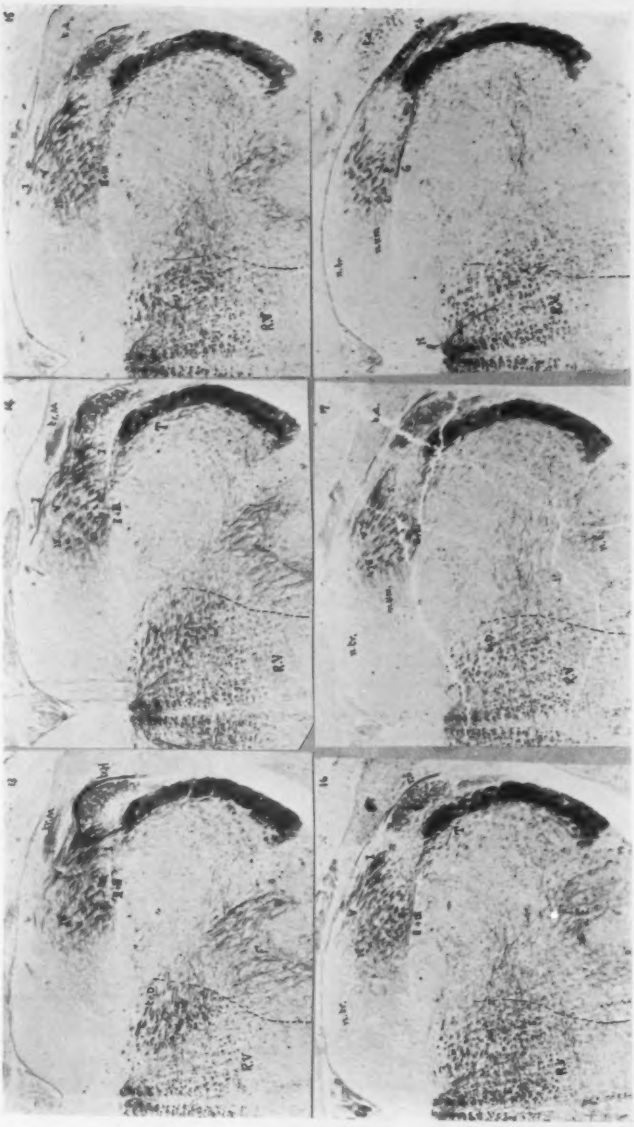
saccular macula not only have no connection with the hearing centers, but distribute themselves in a manner extremely similar to that of the fibres from the utricular macula, only the second alternative would be possible and the problem would have to be stated as follaws: "The saccular macula has a function similar to that of the utricule, and it is necessary to find out methods to detect the static disturbances following its extirpation; a hearing function cannot be accepted." As a matter of fact, the fibres from the saccular macula (S. later) are distributed in the same nuclei as are the fibres from the utricle and from the semicircular canals and have nothing in common with the primary cochlear centres.

The first step in determining the central "projection" of the labyrinth was, of course, to "project" the labyrinth end-organs in the ganglion of Scarpa. This step has already been taken. As long ago as 1901 Alexander,[1] in spite of the rather poor histological methods available at that time, was able to state that in the ganglion the cells corresponding to the individual end-organs are not mixed up, but the branches for each organ arrive at different regions of the ganglion. In Alexander's paper, the projection of the saccular macula and of the crista of the posterior canal were not accurately determined. Cajal,[9] with birds, without entering into details, also announced the existence of an anatomical projection. Beccari,[3] with reptiles, using the Cajal silver method, gave an accurate description, but as to the labyrinth in mammals, nothing was established until recently.[30, 32] Using a very extensive material (embryos of mouse and cat and young mice) and a special modification of the Cajal method, it was possible to determine completely the course of the peripheral branches of the vestibular nerve in the interior of the ganglion and thus establish that each end-organ is projected in a special region of the ganglion of Scarpa. Fig. 14 shows this projection. Studies were also made to determine how the fibres arising from the different regions of the ganglion constitute the bundles of the central division of the vestibular nerve.[30, p. 37; 32, p. 198]

The next step was to follow these bundles to their endings in the nuclei in the floor of the fourth ventricle; this has now been accomplished.

2. Historical.

The central distribution of the vestibular nerve has been repeatedly studied (S. literature by P. v. Gehuchten[15] or Marburg[33]) but most of the authors have used the Marchi or the Weigert method and, therefore, have been unable to determine the behavior of the individual fibres, or even bundles of fibres. The Weigert method



Figs. 27-32.

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stains only the myelinated part of the nerve fibres and, therefore, does not give any information about their divisions or their collaterals. The Marchi method stains only the degenerated myelin fibres. Consequently, the ideal procedure would be to cut the central branch of the nerves from the individual end-organs and stain the degenerated fibres by the Marchi method. Unfortunately, it is impossible thus to obtain satisfactory results.

When the labyrinth end-organs are destroyed the vestibular nerve does not degenerate—at least not regularly—because the neurones in the ganglion of Scarpa remain untouched, and if the ganglion of Scarpa is destroyed the whole vestibular nerve (together with the also destroyed cochlear nerve) degenerates. When the whole eighth nerve is degenerated it is totally impossible to recognize the individual bundles; such preparations are even less useful, for this purpose, than the Weigert specimens. This is why the attempt of Winkler to determine in Marchi preparations the course of the individual bundles of the eighth nerve, after extirpation of the internal ear, was a failure, and the description he gave a purely hypothetical interpretation.*

If exception is made of the very fragmentary details given by Kölliker[27] and Held[19] it can be said that the first and, until now, only description of the central distribution of the individual vestibular fibres in mammals was given by Cajal in 1896.[7, 10]

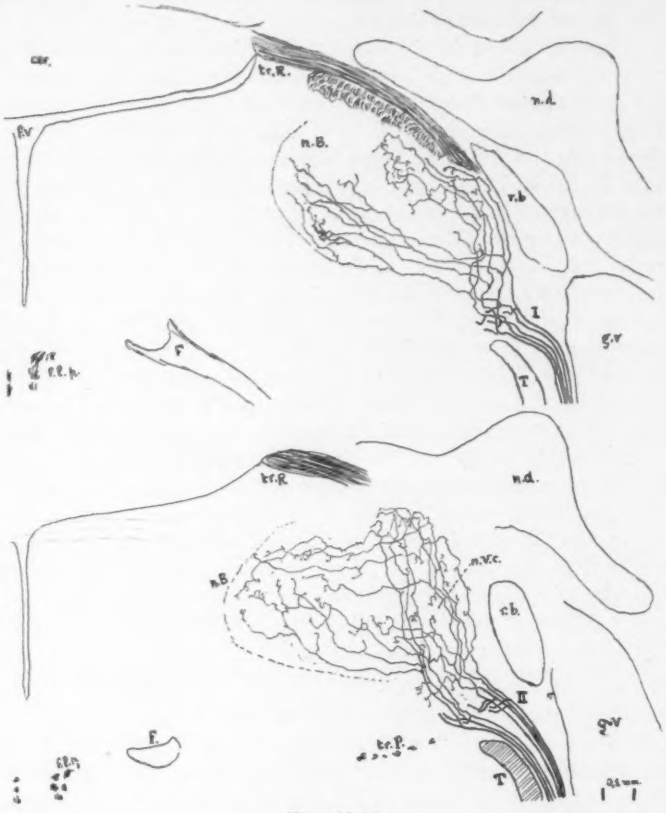
With the mouse, using Golgi stains, Cajal found that the vestibular fibres after their arrival at the floor of the fourth ventricle divide into two branches; one ascends towards the cerebellum, where it penetrates; during its ascending course it gives collaterals in the vestibulo-cerebellosus and in the Bechterew nuclei; the other branch descends in the classic spinal root of the vestibular nerve; it gives collaterals in Deiter's nucleus, the nucleus proper of the descending root, in the nucleus angularis and the nucleus triangularis. Cajal considered the vestibular nerve as homogeneous and described only one class of fibres. These apparently correspond to our Group II and, therefore, are fibres from the semicircular canals (S. later).

3. Materials and Methods.

Since the experimental method (Marchi) has proved to be unreliable for the study of the central projection of the labyrinth, an attempt has been made to solve this question with the help of the anatomical methods in the most suitable material (mice and cats).

*We cannot refer here to the very important papers of Grobbels(16) on the vestibular nerve in birds, because the distribution of the vestibular nerve in mammals and in birds seems to be quite different and the homology of the cell nuclei has not been established yet.

The Golgi method has been used in young mice and cats for determining the existence of different classes of fibres in the central vestibular root, and the distribution of each class of fibres. Since all the fibres have partly common distribution, very extensive material (over a thousand brains) had to be used, because it was necessary



Figs. 33-34.
See legends at end of article.

to have preparations in which only fibres of one class were stained. This has demanded very numerous trials; as a matter of fact, this study was begun in 1922, and although it soon was found that different categories of fibres were present, to determine the characteristics of each of them and their peripheral origin has been much

more difficult, owing to the fact that in the vestibular nuclei besides the vestibular fibres many other pathways end (from the cerebellum, from the restiform body and from the reticular substance) and, therefore, it was necessary to determin the course and ending of every kind of fibre.

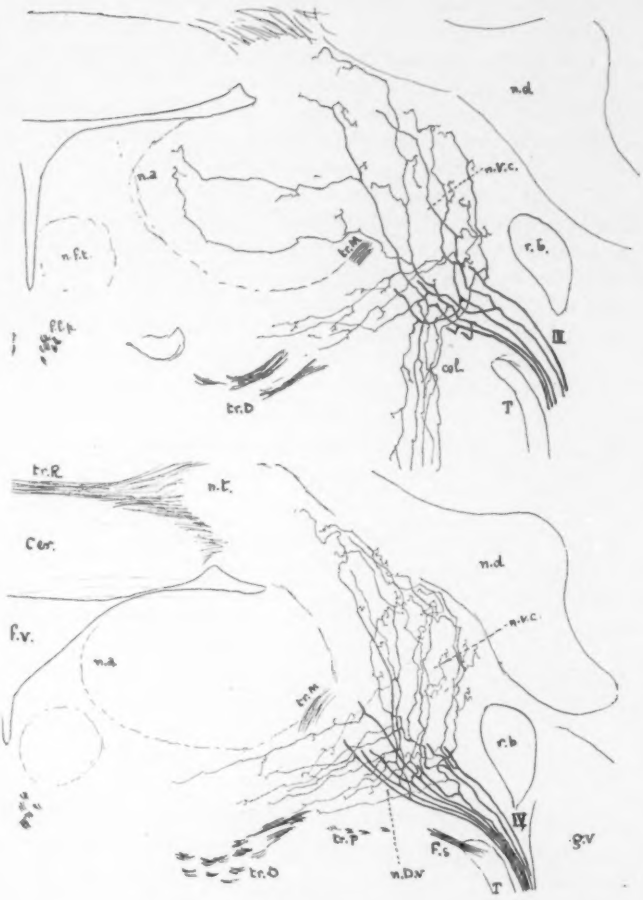
The Cajal silver method with mouse and cat embryos has been very useful for determining the origin in the ganglion of Scarpa of the bundles found in the Golgi stains. The Weigert-Kulschitzky method, used in Flechsig's myelinization method, has been extremely useful to differentiate the vestibular fibres from the other pathways ending in the nuclei in the floor of the fourth ventricle. Bechterew found that the vestibular fibres become myelinated at earlier stages than do the cochlear fibres; and elsewhere[30] it was established that the fibres from the different labyrinth end-organs begin the myelinization process at different times. The Weigert method then only stains only one or several of the bundles of the vestibular nerve to be described later. On the other hand, since the vestibular fibres become myelinated earlier than the cerebellar ones, by choosing animals of appropriate age it is possible to have stains of only vestibular fibres. For instance, in Figs. 15-32 the vestibular fibres already are myelinated, whereas most of the cerebellar fibres still have no myelin and, therefore, remain unstained.

In order to follow the bundles of fibres more easily and more accurately, reconstructions on glass plates of the nerve and its branches have been made, upon which the whole course of the different fibre bundles from the end-organs, through the ganglion of Scarpa into the floor of the fourth ventricle appears. Upon these reconstructions the facial and intermediate nerves also can be studied.

The following description will be made on the basis of drawings and photographs taken from preparations from mouse-brains; but with slight modifications it is also valid for the rabbit and cat.

4. The Central Distribution of the Vestibular Nerve.

The general distribution of central division of the vestibular nerve can be seen in Figs. 15-32 which reproduce photographs of the vestibular region of the medulla of a seven-day-old mouse stained after Weigert-Kulschitzky. At this age the vestibular nerve is completely myelinated while the cerebellar fibres still are almost unmyelinated; only the first or vertical part of the tract of Russell and a few of the fibres descending from the cerebellar cortex in their vertical course towards the floor of the fourth ventricle take the hematoxylin stain. Therefore, in the ascending and descending roots of the vestibular nerve there are practically only vestibular fibres.



Figs. 35-36.

See legends at end of article.

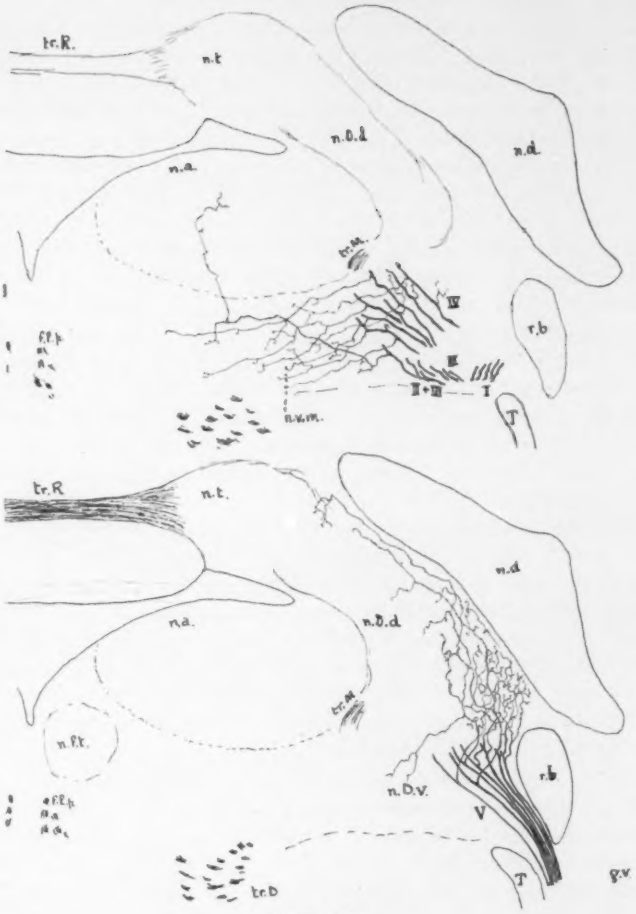
The vestibular nerve enters the medulla between the descending trigeminal root and the primary cochlear nuclei and penetrates into the nuclei in the floor of the fourth ventricle between the trigeminal root and restiform body. Shortly after their arrival at these nuclei, all the vestibular fibres, without exception,* divide into two branches —one that ascends towards the cerebellum, but without entering it (S. later), and another that descends towards the spinal cord. The ascending branches are more or less parallel to the restiform body and, therefore, may be contained almost totally in a single cross-section of the medulla; the descending branches are more or less parallel to the longitudinal axis of the medulla and, therefore, only a small part of them appear in each cross-section (see Figs. 15-32). If it is desired to have the whole descending branch in a single histological section, it is necessary to make longitudinal or horizontal sections of the medulla. The reader who wishes more information about the general distribution of the vestibular fibres may consult the papers of Cajal, Winkler, Leidler, v. Gehuchten or the important monograph of Fuse. To these descriptions must be added the following details:

The vestibular fibres do not all distribute themselves in the same way. They may be divided into five groups having quite definite characteristics.

Group I (Fig. 33). The fibres of Group I enter the medulla first; among them there are only thin and medium fibres. They divide immediately into ascending and descending branches. The ascending branches penetrate the oral part of the vestibulo-cerebellosus nucleus where they end by means of a rather simple ramification, and in addition they give off collaterals for the nucleus of Bechterew. The descending branches remain, during their early course, in the angle between restiform body and trigeminal root; they will later constitute the most lateral bundles of the vestibular descending root (see Fig. 37).

Group II (Fig. 34). The fibres of Group II are thicker than those of Group I. They enter into the nuclei of the fourth ventricle a little caudal and medial to Group I and divide immediately into ascending and descending branches. The ascending branches are very similar to those of Group I; but the descending ones, as we shall see later, are somewhat different; at the beginning they place

*On the basis of Weigert's or Marchi's stains, several authors have expressed the belief that the caudal entering fibres do not give off ascending branches; since these methods do not stain the (unmyelinated) part of the fibre in which the division takes place, it is evident that these statements were hypothetical.



Figs. 37-38.

See legends at end of article.

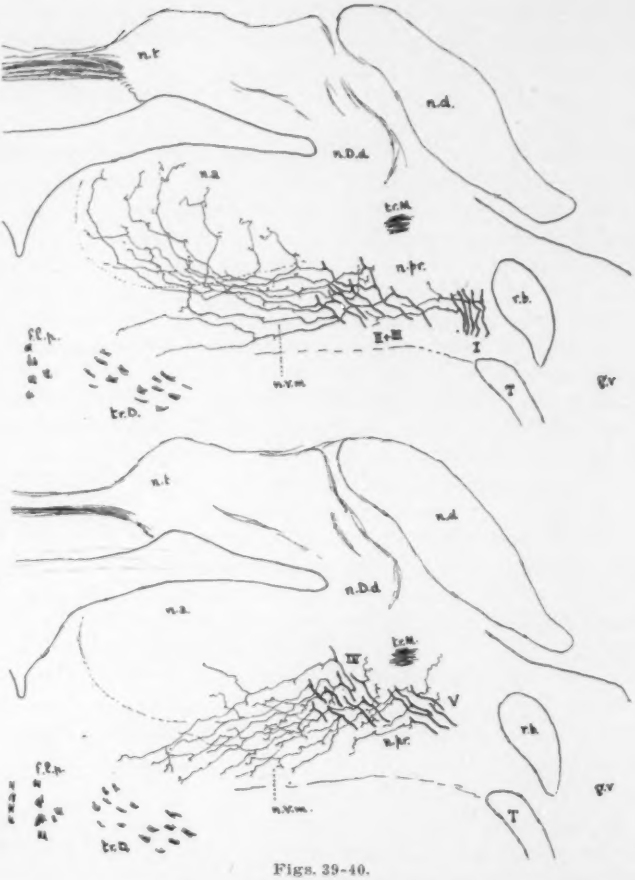
themselves close to the trigeminal root, medial to the descending fibres of the Group I (see Fig. 37, II and III).

Group III (Fig. 35). This group contains thick and medium fibres. They enter caudal to Group II, and are characterized by their giving off a collateral branch that goes into the substance passing immediately medial to the sensory nucleus of the trigeminal nerve. These collaterals (see Fig. 35, col.) arise either from the ascending or from the descending branches. Their terminations will be described in another paper. The ascending branches of Group III sometimes are similar to those of Group II and sometimes to those of Group IV, but always give off collaterals for the ventral Deiter's nucleus, which fibres of Groups I and II do not do. The descending branches constitute parallel bundles that partly are mixed with those of Group II, but chiefly are placed a little dorsal and lateral to them (see Fig. 37, II and III).

Group IV (Fig. 36). This group contains thick and medium fibres and a few thin ones. They enter into the floor of the fourth ventricle caudal and lateral to those of Group III. The division does not take place immediately but in the interior of the ventral Deiter's nucleus. The ascending branches do not give any collateral to the nucleus of Bechterew, but only to the vestibulo-cerebellosus nucleus. The descending branches constitute parallel bundles in the dorso-medial region of the descending vestibular root (see Fig. 40, IV).

Group V (Fig. 38). Finally Group V contains chiefly medium fibres and only a few thin ones. They are the most caudal and lateral fibres of the vestibular nerve. The division takes place after the fibres have traversed a relatively long way in the floor of the ventricle. The ascending branches give off collaterals to the latero-caudal part of the vestibulo-cerebellosus nucleus; the descending ones constitute parallel bundles in the dorsolateral region of the descending spinal root (see Fig. 40, V).

A few words about the nuclei where the ascending branches end will provisionally complete this description. In the vestibulo-cerebellosus nucleus (or better said, complex of nuclei) several regions have to be differentiated. In the first place, there is a region, the dorso-caudo-medial one, inhabited by big cells, sometimes called the dorsal Deiter's nucleus, in which no vestibular fibres penetrate; if these cells have direct connection with the vestibular nerve, it is by means of only a few thin collaterals. This is chiefly a cerebellar nucleus. The part of the vestibulo-cerebellar complex in which the ascending branches of Groups IV and V penetrate has quite a different structure from that of the oral part where the fibres of



Figs. 39-40.

See legends at end of article.

Groups I and II end; but the fibres of Groups IV and V, and possibly also of III, often give off one or several collaterals that go forward and end in this oral part of the vestibulo-cerebellar complex: No collateral of the fibres of Groups I and II seem to go backwards to the central and lateral parts of the vestibulo-cerebellar complex where the fibres of Groups IV and V give rise to their end ramifications. The cell mass called the nucleus of Bechterew is continuous with the nucleus angularis. In this nucleus, chiefly in its central part, the fibres of cerebellar origin are more numerous than the vestibular fibres.

Whether or not the ascending branches of the vestibular fibres reach the cerebellar cortex is a question that I cannot answer with absolute certainty. It is certain that while they are traversing the vestibulo-cerebellar complex all of the vestibular fibres give off numerous collaterals and soon become very thin, so that they may not go very far, and it is sure that many end there, but on the basis of Golgi stains the possibility of the existence of a few thin collaterals entering the white cerebellar substance and afterwards reaching the cerebellar cortex cannot be excluded. I never saw, in many hundreds of well-stained preparations, a single vestibular fibre entering the cerebellum; all had their endings in the vestibulo-cerebellar complex; the fibres of Groups IV and V (see Figs. 36 and 38) are those that go farthest upwards because they give off terminal fibres for the superior part of the vestibulo-cerebellar complex, which lies in contact with the nucleus tecti, but I never saw them going into the white cerebellar substance. It must then be stated that a direct connection of the vestibular fibres with the cerebellar cortex is extremely doubtful, and if present it is only by means of few, thin collaterals. With the help of the Marchi method, investigators have arrived at conflicting results (S. literature by Ingvar,[24], Herrick[23] or Ariens Kappers.[26])

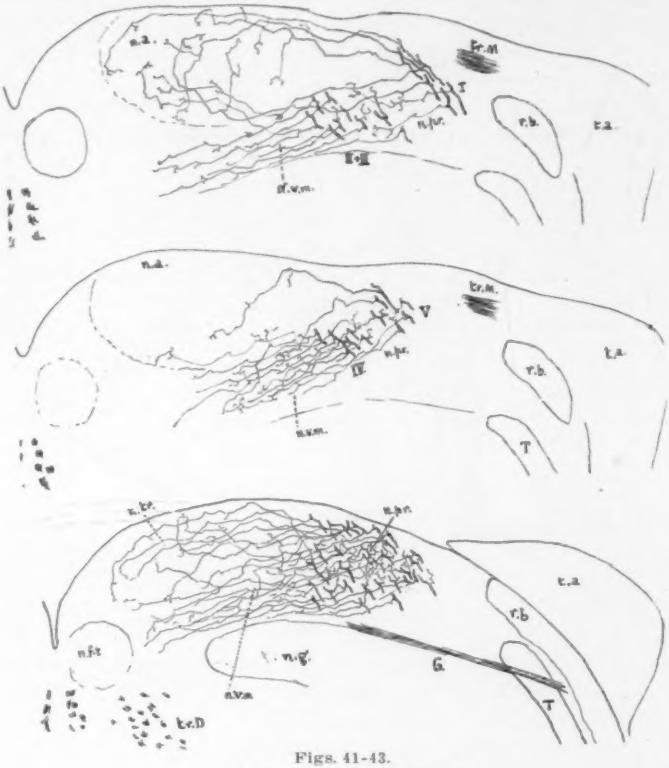
The course of the descending branches of the five groups between the entrance of the nerve and the posterior level of the tuberculum acusticum can be seen in Figs. 37, 39-43.

The descending fibres of Groups I and II establish similar connections; they first lie in the angle between trigeminal root and restiform body, and as long as the other groups of fibres are entering they do not give off collaterals. When Group V enters, they bend, Group I lateral and Group II medialwards, Group II constituting the more ventral bundles of the descending spinal root.

As soon as Group V has entered the medulla, Group II begins to give off collaterals, short ones that remain in the nucleus proper

of the descending root and long ones that go medialwards pass through the nucleus ventro-medialis and finally turn upwards in graceful arcs and penetrate into the nucleus angularis, where they end by means of a rather extensive ramification.

The fibres of Group I have all their collaterals going medialwards; some of them are short and end in the nucleus proper of the



Figs. 41-43.
See legends at end of article.

spinal root and nucleus ventro-medialis; but other long and thick collaterals go upwards and enter the nucleus angularis at its caudal and dorsal boundary. After the entrance of these collaterals the angularis nucleus ends and the triangularis one begins.

The descending fibres of Group III run in parallel bundles, a little dorsal and lateral to Group II. They give off collaterals to the ven-

tral Deiter's nucleus, *i. e.*, between the entering root fibres of Groups III, IV and V, and then numerous ones to the nucleus ventro-medialis, and a few to the nucleus angularis, in a manner similar to that of the fibres of Group II.

The fibres of Group IV constitute the dorso-medial bundles of the descending root; they give off collaterals to the ventral Deiter's

Fig. 44.
See legends at end of article.

nucleus, nucleus proper of the descending root, and nucleus ventro-medialis; they do not give off collaterals for the nucleus angularis, only a few thin branches arising from their collaterals for the nucleus ventro-medialis seem to go into the ventral part of the angularis nucleus.

The fibres of Group V run in the dorso-lateral bundles of the spinal root. They give off collaterals to the nucleus proper of the

descending root and to the nucleus ventro-medialis; only very few branches enter the nucleus angularis.

When the nucleus angularis ends and the nucleus triangularis begins, *i. e.*, at the level of the caudal and of the tuberculum acusticum, the different groups of fibres cease to show individual characteristics; they retain approximately the same relative position in the descending spinal root but all have very much the same distribution. All of them give collaterals to the nucleus proper of the descending root, the nucleus triangularis and the nucleus ventro-medialis. In other words, all the five groups of fibres have approximately the same connections in the caudal third of the vestibular nuclei. This is why in Fig. 43 fibres from all the five groups have been drawn together.

5. The Peripheral Origin of the Five Groups of Vestibular Fibres.

If the bundles of fibres are followed in series of sections through the medulla and labyrinth from mouse or cat embryos, stained after Cajal, or from young mice (three to six days) stained after Weigert-Kulschitzky, or better, if reconstructions of the bundles are made on glass plates, it is found that each of the five groups of vestibular fibres arise in a different region of the ganglion of Scarpa.

Groups I and II arise from the regions of the ganglion where the fibres of the semicircular canals have their cells of origin (see Fig. 14). As shown in another paper,[30] (see Fig. 5); [32] (see Fig. 82) the cells for the anterior and external cristae lie in the anterior and dorsal part of the ganglion, whereas the cells for the posterior crista lie in the dorso-caudal region, but the fibres from the three semicircular canals come together when they start their way toward the medulla and constitute a single bundle, which contains Groups I and II of root fibres. Since the fibres of Group I are thinner than those of Group II, there can be little doubt that Group I contains the fibres from the peripheral parts of the cristae, whereas Group II contains the fibres from the central parts of the cristae.

The fibres from the utricular and anterior saccular nerves enter the medulla a little more caudal than those of the semicircular canals; they undoubtedly constitute Group IV, but whether Group III is a part of the utricular nerve (region *b* of the macula) or a part of the nerves for the cristae of the semicircular canals (thick fibres from the centrum of the cristae) I have not been able to determine. This question, however, is only of secondary importance because the region *b* of the utricular macula has a structure very similar to that of the central regions of the cristae, and Group III has common characteristics with Groups II and IV.

The fibres from the posterior saccular nerve constitute Group V; whether or not a part of them run in Group IV, I cannot tell, but this question has, of course, no importance because Groups IV and V have very similar distribution and Group IV contains the fibres from the anterior saccular nerve.

Since the anterior saccular nerve forms a part of Group IV and the posterior saccular nerve forms Group V, it is absolutely certain that *the saccular fibres do not join the cochlear nerve*, and there cannot be the slightest doubt that *the saccular macula is represented in the medulla in the same nuclei as the utricular macula, and the semicircular canals.*

Since this conclusion has physiological significance, it is worth while to reproduce in Fig. 44 the whole course of the posterior saccular nerve, from the macula, through the ganglion of Scarpa, to the medulla. In another paper,[30, 32] it was already established that the anterior saccular nerve enters the ganglion in its anterior part, but runs backwards to join the posterior saccular nerve in the interior of the ganglion, so that the whole macula sacculi is represented in the ventro-caudal region of the ganglion of Scarpa. From this region arise the caudal vestibular fibres which constitute Group V and the dorsal fibres of Group IV.

6. Discussion.

With the foregoing description the projection of the labyrinth in the primary vestibular nuclei has been established as accurately as the neuro-anatomical methods of today allow. But with this only a very small step towards the complete knowledge of the anatomy of the vestibular system has been made. The next step must be to determine the origin and course of the different collateral or terminal pathways that, coming from other centers, enter the primary vestibular nuclei; then it will be necessary to study the different types of cells that inhabit the primary vestibular nuclei and the pathways arising from them. In the literature there already exists some information but it is undoubtedly insufficient. As a matter of fact when one tries to determine the pathways that close the vestibular reflex arc, on the basis of the available data, the result is very discouraging.[32, p. 207]

Although without a description of the terminal nuclei it is impossible to give complete details, the foregoing description enables us to state that the semicircular canals and the maculae have partly different, but also partly common, representation in the nervous system, and that both maculae have similar representation, totally dif-

ferent from that of the cochlear nerve. It is evident that the macula sacculi must have a function similar to that of the other labyrinth organs and totally different from the cochlear function. On the other hand, it cannot be accepted that the maculae and the semicircular canals may have quite different functions or work independently; *the whole static labyrinth must be considered more or less as a unit because its centres in the medulla are a unit.* The results of physiological experiments reinforce this conclusion.[21a, 22]

It has been accepted that the fibres from the three semicircular canals have a different course in the medulla (Jones[25]). This cannot be supported by the anatomical findings. All the semicircular fibres are parallel and the fact that they constitute two groups (I and II and perhaps also a part or the whole of Group III), does not signify that each group corresponds to a canal, but that Group I contains thin and medium fibres and Group II medium and thick fibres from all of the canals. On the other hand, the semicircular canal fibres travel through the whole length of the descending root so that each canal is represented in every part of the vestibular nuclei. The same happens with the maculae. The results of physiological experiments reinforce this conclusion.

7. Conclusions.

1. The fibres from the labyrinth end-organs (cristae and maculae) have been followed from the labyrinth, through the ganglion of Scarpa, into the medulla oblongata.

2. The central division of the vestibular nerve may be divided into five groups of fibres, each having special distribution.

3. Groups I and II contain fibres from the semicircular canals; Group IV fibres from the utricular macula, and Group V fibres from the saccular macula; it has not been determined whether Group III contains fibres from the central part of the cristae or from the region *b* from the utricular macula. Both alternatives are possible because all these fibres are thick.

4. The semicircular canals and the maculae have partly different and partly common central representation. The fibres from both maculae have extremely similar distribution.

5. The whole static labyrinth must be considered as a physiological unit; the saccular macula must, of course, have a function similar to that of the rest of the labyrinth; an acoustic function for it cannot be accepted, because it has connections with the centres and pathways involved in the regulation of equilibrium, but no connection with the pathways and centres involved in hearing.

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Fig. 1. Longitudinal section through the primary acoustic nuclei of a four-day-old cat. Method of Golgi.

G. v.—ganglion ventrale, with I, II, III its three regions of specific structure; p. n. (a) and p. n. (b)—posterior nucleus with its two main regions; the section goes through the lateral region (b) of that part of the posterior nucleus where the fibres from the apex of the cochlea give off collaterals, and through the medial region (a) of that part of the posterior nucleus where the fibres from the central part of the cochlea give off collaterals. T. a.—Tuberculum acusticum. I, II, III—its three regions of specific structure.

N. c.—Fibres of the cochlear nerve with ascending or anterior branches (a) for the ganglion ventrale and descending or posterior branches (p) for the tuberculum acusticum and posterior nucleus. C. f.—centrifugal fibres, i. e., fibres coming from higher nuclei, for the tuberculum acusticum.

This figure shows that each cochlear fibre establishes connections with many categories of nerve cells; therefore, the impulses produced in the primary acoustic nuclei will not correspond exactly to those set up in the cochlea, but rather will be the result of the activity of the cells inhabiting these nuclei. In another paper the description of the fibres and cells will be given.

Fig. 2. Diagrammatic representation of the projection of the ganglion of Corti in the primary acoustic nuclei.

I. Projection of a longitudinal section of the primary acoustic nuclei on a longitudinal plane.

N. c.—cochlear fibres, the numerals 1-17 indicate their origin in the different regions of the ganglion of Corti; fibre 1 comes from the basal and fibre 17 from the apical region of the ganglion of Corti. G. v.—ganglion ventrale; T. a.—tuberculum acusticum.

II. The broken line represents the arc line formed by the division points of the cochlear fibres when projected in a longitudinal plane (Diagram 1). G. c. represents an uncoiled ganglion of Corti projected on that line. A—apical, C—central and B—basal parts of the ganglion. The contours of the ganglion have been drawn, taking in account the data on the number of cells per unit of length of organ of Corti published by Guild and coworkers.

Figs. 3-12. Serial sections through the cochlea and medulla oblongata of a 12-day-old mouse. Method of Weigert-Kultchitzky. These drawings are intended to give an approximate idea of the twisted course of the cochlear nerve. The bundles of fibres arising from the different parts of the ganglion of Corti have been marked with the numerals 1/4, 1/2, 2/2, 3/2, 4/2 (a and b) which permits of following them from the ganglion through the changes in relative position, to the medulla.

G. v.—ganglion ventrale with the cochlear fibres stained from their origin to their points of division; the ascending and descending branches still are almost unmyelinated and, therefore, do not complicate the microscopic image. G. c.—ganglion of Corti; n. a. p.—nervus ampullaris posterior; G. s.—ganglion of Scarpa.

The sections are some 25 microns thick; beginning with the section when the caudal end of the cochlea appears the number of the reproduced sections are 3, 5, 7, 8, 10, 14, 16, 18, 20, 22.

Fig. 13. Cross-section through the head of a cat embryo in which the organ of Corti has not yet been penetrated by the peripheral branches of the cochlear nerve. Cajal's silver method. C. l.—capsule of the labyrinth; fac.—facialis nerve; A. e.—nerve of the external ampoule; M. u.—macule of the utricule with b. c regions of different innervation; M. s.—macule of the saccule; G. S.—Scarpa's Ganglion; c. rest.—restiform body; Vest.—spinal root of the vestibular nerve; VII—sensory branch of the VIIth nerve; V—descending root of the Vth nerve.

G. C.—Corti's Ganglion; N. c.—cochlear nerve; r. d.—descending or posterior divisions branches of the cochlear fibres ending in the tuberculum acusticum, Tub. ac.

Fig. 14. Diagram indicating the projection of the labyrinth end-organs in the ganglion of Scarpa. A. c.—anterior crista; A. l.—lateral crista; A. p.—posterior crista; M. u.—macule of the utricule; M. s.—macule of the saccule. G. S.—ganglion of Scarpa; a. and p.—anterior and posterior part of the ganglion. R. u.-a.—Ramus utriculo-ampullaris; R. sac.—Ramus sacularis. From Lorente de No, Ausgewahlte Kapitel, etc., Erg. d. Physiol., 32, 1931.

Figs. 15-32. Photographs of serial sections through the vestibular region of the medulla of a seven-day-old mouse. Method of Weigert-Kultchitzky. The number on top of each photograph is the number of the section in the series beginning with the section reproduced in Fig. 15.

ABBREVIATIONS.

a.—region of the reticular substance where homolateral fibres from the vestibular nuclei form ascending and descending pathways.
Ab.—abducens nerve.
cer.—cerebellum.
c. f.—fibres from the cerebellar cortex for the vestibular nuclei.
col.—collaterals from the fibres of the IVth vestibular group.
F.—facialis nerve
F. s.—sensory facialis nerve.
f. v.—fourth ventricle.
G.—glosso-pharyngeus nerve.
g. v.—ganglion ventrale (cochlearis).
H.—hypoglossus nerve.
n. a.—nucleus angularis.
n. B.—nucleus of Bechterew.
n. D. d.—dorsal division of the nucleus of Deiters.
n. D. v.—ventral division of the nucleus of Deiters.
n. d.—nucleus dentatus.
n. f. t.—nucleus funiculi teretis (a part of the vestibular system).
n. g.—nucleus of glosso-pharyngeus nerve.
n. pr.—nucleus proper of the descending vestibular root.
n. t.—nucleus tecti.
n. tg.—sensory nucleus of trigeminus nerve.
n. v. c.—nucleus vestibulo-cerebellosus.
n. tr.—nucleus triangularis.
n. v. m.—nucleus ventro-medialis.
R. T.—reticular nuclei belonging to the trigeminus system.
R. V.—reticular nuclei belonging to the vestibular nerve.
r. b.—restiform body.
T.—spinal root of the trigeminus nerve.
t. a.—tuberculum acusticum (cochlearis).
tr. D.—tractus of Deiter's.
tr. H.—acoustic tract of Held.
tr. M.—acoustic strias of Monakow.
tr. P.—tractus of Probst.
tr. R.—tractus of Russell.
tr. s.—tractus solitarius.
Vest.—vestibular nerve with I, II, III and IV and V, its five bundles.

In these figures the fundamental factors of the vestibular system are contained.

In the vestibular nuclei at the floor of the fourth ventricle in addition to[1] the vestibular fibres (Figs. 15-32, Vest.) enter[2] the tract of Russell (Figs. 15-17, tr. R),[3] fibres from the cerebellar cortex (Figs. 19-26, c. f.), and collaterals from[1] the restiform body,[2] the reticular substance (R. V.),[3] fasciculus longitudinalis posterior (f. l. p.) and[4] tractus predorsalis which in part pass through the nucleus funiculi teretis (n. f. t.) and partly are mixed with efferent pathways form the angularis and ventro-medialis nuclei. These collaterals are not yet myelinated. The different bundles of vestibular fibres can be seen in all the figures indicated with the numerals I to V. Figs. 33-43 indicate more completely the course of these fibres as it appears in Golgi stains.

The vestibular nuclei have been provisionally marked with the following names: nucleus of Bechterew, nucleus vestibulo-cerebellosus, nucleus angularis, nucleus triangularis, nucleus ventro-medialis, dorsal division of the nucleus of Deiter's—which is not a vestibular nucleus, ventral division of the nucleus of Deiter's, and nucleus proper of the descending root. A further subdivision and the description of each nucleus will be made in another paper.

Some of the efferent pathways taking origin from the vestibular nuclei already are myelinated and can be seen in the photographs. The caudal part of the tr. vestibulo-mesencephalicus appears in Fig. 15. The descending tractus of Deiter's begins to appear in Fig. 16; in Fig. 20 it has already reached the reticular substance, later it will place itself in the lateral part of the tr. predorsalis (see Fig. 32).

A very important, apparently unknown detail is that the fibres of the descending tract of Deiter's give off numerous collaterals to the reticular nuclei.

The fibres for the f. l. p. arising chiefly from the nucleus ventro-medialis can be seen in Figs. 17-22. In more caudal sections these fibres are still unmyelinated.

The powerful systems of fibres for the reticular nuclei can be seen in Figs. 17-22; they constitute ascending and descending pathways in the region marked with "a" in Fig. 17. In more caudal levels (Figs. 22-32) these fibres are mixed with those going towards the f. l. p.; they do not appear in the photographs because they still are unmyelinated.

The dorsal part of the reticular substance belonging to the vestibular system (V. R.) is enclosed in all the figures between the raphe and the broken ink line. In Fig. 15 the powerful tracts that connect the reticular substance of the medulla with that of pons, midbrain and thalamus cross the fibres of the VIth nerve. The already myelinated tracts are chiefly de-

scending; in Fig. 32 these tracts have joined the tractus predorsalis; they give off numerous collaterals for the reticular nuclei and also, as already mentioned for the vestibular nuclei.

Each of the regions of the reticular substance has an especial structure. At the level of Fig. 16 a change in structure appears and again another change at the level of Fig. 24. The crossed efferent pathways of the medial reticular nucleus (enclosed between tr. predorsalis and tr. of Deiters) appear clearly in Figs. 22-31; caudal to Fig. 32 the medial region of the reticular substance has again different structure and connections.

In previous work (31a, 32) the importance of the reticular nuclei for the production of the vestibular reflexes has been demonstrated with physiological experiments. Anatomically the pre-eminent role of these nuclei is evident, because they receive from and send to the nuclei at the floor of the fourth ventricle powerful tracts, bigger than the f. l. p., which, on the other hand, gives numerous collaterals for the reticular substance and after having innervated the motor nuclei of the eye muscles ends in the thalamic reticular nuclei.

Figs. 33-43 reproduce the course and distribution of the five bundles of the vestibular nerve. Since the reproduction of Golgi preparations would demand very numerous illustrations and besides would make very difficult for the reader the identification of the bundles in the Weigert's series of adult animals, the course of the fibres has been indicated in diagrams having the contours of Figs. 15-32 (Fig. 33=17; 34=18; 35=19; 36=21; 37 and 38=23; 39 and 40=24; 41 and 42=28; 43=32). This has been made, of course, after careful comparison of series stained after Golgi, Weigert, Cajal and Cox.

The abbreviations are the same as in Figs. 15-32.

Fig. 33. Entrance of the fibres of Group I.

Fig. 34. Entrance of the fibres of Group II.

Fig. 35. Entrance of the fibres of Group III.

Fig. 36. Entrance of the fibres of Group IV.

Fig. 37. The descending branches of Groups I, II, III and IV. In the angular nucleus the collaterals of the descending branches of the Groups I, II, III.

Fig. 38. Entrance of Group V.

Fig. 39. The descending branches of Groups II, III and IV.

Fig. 40. The descending branches of Groups IV and V.

Fig. 41. The descending branches of Groups I, II and III.

Fig. 42. The descending branches of Groups IV and V.

Fig. 43. The descending branches of all the five groups at the level of the posterior end of the tuberculum acusticum.

Fig. 44. Cross-section through the head of a mouse embryo of some 14 m.m. length. Cajal's silver method.

The section shows the whole course of the posterior saccular nerve from the macula through the ganglion of Scarpa to the medulla. B.—Capsule of the labyrinth. M. s.—Macule of the saccule; G. S.—Ganglion of Scarpa; Vest.—the saccular fibres of the vestibular nerve entering the medulla. d. v. VIII, the descending branches of the oral entering vestibular fibres (Groups I-IV); note that the branches of Group I cross the entering saccular fibres in order to place themselves on the inside of the restiform body. G. v.—ventrale nucleus of the cochlear nerve with the anterior or ascending divisions branches in cross-section; T. b.—trapezoid body; R. b.—restiform body; n. D. v.—ventral part of Deiter's nucleus; n. D. d.—dorsal part of Deiter's nucleus; c. f.—descending cerebellar fibres that constitute in the vestibular nuclei several systems of fibres running parallel to the vestibular ones. IV. V.—fourth ventricle. T. d. d.—Tractus Deiter's descendens; f. l. p.—secondary vestibular fibres in their way towards the posterior longitudinal bundle. X1, X2, X3; three bundles of the facialis nerve; X1 is the motor one, X2 and X3 belong to the autonomic system.

Note that the ventral nucleus (G. v.) of the cochlear nerve still lies in the dorsal part of the medulla; in later stages of development it will descend to the place it occupies in Figs. 34-41, thus modifying the position of the different structures in the lateral part of the oblongata. This change in the form and position of the acoustic nuclei, together with the migration of Scarpa's and Corti's ganglions represent the mechanical cause of the appearance of the so-called Oort's anastomose between the cochlear and vestibular nerve, which, as described elsewhere,[30 32] is not real anastomose but is formed by cochlearis fibres that, having a somewhat abnormal course, seem to join the vestibular nerve.

FREE IODINE IN THE TREATMENT OF VASOMOTOR RHINITIS AND THE SYMPTOM COMPLEX OF SNEEZING AND NASAL HYDRORRHEA — PRELIMINARY REPORT.

Dr. Morris Levine, Brooklyn.

This report is based on treatment of a limited series of cases of vasomotor rhinitis, common cold and hay fever with subcutaneous injections of a 5 per cent sodium iodide solution containing traces of free iodine. The encouraging results so far obtained justify further clinical research concerning the value and limitations of this preparation.

Although of diverse etiology, vasomotor rhinitis, common cold and hay fever have one underlying pathological finding in common: a waterlogged condition of the nasal mucous membrane. This accounts for the manifestations of sneezing, nasal stuffiness and profuse watery discharge from the nose in all three diseases.

Pharmacologically, it is established that iodine is eliminated in part by the mucous membrane of the upper respiratory tract, upon which it has a definite stimulating action. With administration of excessive amounts of iodine, irritation takes the place of stimulation, producing coryza, headache, conjunctivitis, laryngitis and bronchitis. From the prompt relief afforded in cases of nasal hydrorrhea, whether due to infection or allergy, it would appear that its influence is principally upon the aqueous metabolism to relieve a waterlogged condition.

Iodine has been used as a folk remedy for colds. In Argentina and Siberia the peasants have depended on a draft containing one drop of tincture of iodine as a means of aborting colds. This treatment has been employed on a wide scale with varying degrees of success.

In considering the applicability of free iodine for nasal hydrorrhea, I was influenced by highly successful results which I have obtained in the use of iodine powder for clearing up the discharge of chronic purulent otitis media.

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My first use of free iodine in sodium iodide solution* was in several refractory cases of vasomotor rhinitis. One of my early patients was a young woman who had been suffering from vasomotor rhinitis for years. Two subcutaneous injections of iodomin dried up her nose perfectly and gave her permanent relief. In another case a man came into my office using a dozen handkerchiefs, so profuse was the watery discharge from his nose. One hour after an injection all discharge had ceased.

To date I have treated approximately twenty cases of pure vasomotor rhinitis and fifty of a mixed type. These included both clinic and private patients. Injections were given every two or three days until the symptoms subsided completely. As a rule, about six injections were required; in the more obstinate cases, as many as ten.

Without exception the treatment gave relief after two or more injections. In some cases improvement was quite striking after the second injection. In others there was a recurrence of symptoms after six months, which, however, was readily controlled by further treatment.

In my experience injections can be given every day without untoward effects. Children three years old have tolerated a full ampoule without harm.

Because of these beneficial results in vasomotor rhinitis, I decided to try iodomin in early cases of common cold before nasal discharge becomes purulent, on the theory that it might bear a favorable influence on the waterlogged condition of the mucous membrane. In a series of approximately twenty cases injections gave early relief of sneezing, nasal stuffiness and watery discharge. Most patients remarked on the ease with which they could breathe soon after an injection, due to shrinkage of the congested membrane. In the majority of cases all symptoms of coryza subsided and did not recur. On the whole, however, results were not quite so gratifying as in vasomotor rhinitis, probably because it is difficult to see patients at the very beginning of a common cold.

Whether iodomin is of value for the purulent stage of rhinitis cannot be answered at this time. I have not as yet studied this phase of therapy sufficiently.

I regard these results as very important, since the complications of common cold are so grave. Cecil found that 46.7 per cent of patients with lobar pneumonia gave a history of preceding cold or

*The preparation employed was Iodomin, supplied by the American Bio-Chemical Laboratories, New York, N. Y. Iodomin is said to be a 5 per cent sodium iodide solution containing traces of free iodine. It is furnished in 1 c.c. ampoules for subcutaneous injection.

cough. I have found the neglected common cold a most prolific cause of sinusitis, tubal catarrh, otitis media, mastoiditis and deafness. It is most unfortunate that every patient with a common cold does not go to bed and call his doctor immediately.

If further clinical study substantiates my results with iodomin injections, we may have at our disposal a most effective therapeutic weapon to fight man's greatest enemy—the common cold.

In approximately ten cases of hay fever injections of iodomin produced shrinkage of the congested nasal mucous membrane and relief from sneezing and watery discharge.

Laryngitis is a definite contra-indication to the use of iodomin, because of the possibility that such patients may develop edema of the larynx on iodine medication. Here it should be used with extreme caution, if at all.

SUMMARY.

Favorable results in vasomotor rhinitis, the early stage of common cold and in hay fever are reported from the use of subcutaneous injections of a 5 per cent solution of sodium iodide containing traces of free iodine. This treatment proves of value wherever the mucous membrane of the nose is waterlogged and appears to act by regulating the water output of the nose. Repeated injections are harmless except in laryngitis.

5022 Fourteenth Avenue.

THE RED SEPTUM.*

Dr. D. C. Jarvis, Barre, Vt.

Introduction: The upper respiratory tract down through the years that have passed has served medical men in various ways as an index to conditions of the body. The condition of the teeth has served as an index to the calcium needs of the body or the ability of the body to utilize calcium. The appearance of the tongue and pharyngeal region has served as an index to the condition of the gastrointestinal tract. The appearance of lymphoid tissue in the upper respiratory tract has served as an index to the need of fat soluble vitamin A. The color changes of the mucous membrane, particularly the mucous membrane of the nose, may serve as an index to the type of preoperative or postoperative treatment best suited to the needs of the patient under consideration.

This index may also be helpful in clinical conditions which are nonoperative. These nasal mucous membrane color changes present a number of patterns to the eye of the observer. One of these patterns deals with the color of the mucous membrane covering the turbinates, more especially the inferior turbinate. Three patterns deal with the degree of pallor of the mucous membrane covering the cartilaginous portion of the nasal septum. Four other patterns deal with the degree of redness of this same mucous membrane. Let us consider the evidence which it is possible to gather in support of this method of determining the type of clinical attack to be employed.

The Nasal Septum: Many times in preoperative or postoperative treatment the question arises as to whether the treatment to be undertaken should be acid or alkaline. As an aid to this decision the nose presents three helpful bits of evidence. The first is the color of the mucous membrane covering the cartilaginous portion of the nasal septum. The second is the color of the nasal discharge, and the third is an associated nasal septum syndrome. On these three is based the decision as to whether acid therapy or alkaline therapy shall be used.

The Red Septum: The mucous membrane covering the cartilaginous portion of the nasal septum presents four shades of red to the eye of the observer. These shades may be designated +1, +2,

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+3 and +4.; +4 is a fiery crimson red, +3 is a crimson red, +2 is a dark red, while +1 is a light red. On the red septum the clinical pictures of the infections are most often built. Clinical observations over a period of 14 years disclose the fact that there is a marked increase in redness of the mucous membrane covering the cartilaginous portion of the nasal septum when the individual selects his food more from acid-ash producing food than from alkaline-ash producing food. In individuals selecting their food more from alkaline-ash producing food than from acid-ash producing food there is an absence of this marked increase in redness. The proof of this is the reduction in the degree of redness of the septal mucous membrane when an increased intake of alkaline-ash food is prescribed, plus the use of alkaline therapy. The time required for the reduction in redness varies from two to six months, depending on the age and weight of the patient. Another proof is the change in color of a pale septum to red when an increased intake of acid-ash food is prescribed, plus the use of acid therapy. The time required for this change varies from 10 days to one month, depending on the age and weight of the patient.

The Red Septum Nasal Discharge: Patients showing a red septum on examination may or may not have an associated nasal discharge. If one is present it is usually due to a crossing of the line of tolerance for acid-ash producing starches. It is characteristic in color, having a yellow tinge. The mucopurulent element of the nasal discharge is due to an excessive intake of starch, but the watery element is due to an excessive intake of sweets. These two together represent a trouble-making combination. The reduction in carbohydrate intake is the most frequent need to be met and produces the most striking results.

The Red Septum Syndrome: Associated with the increased redness of the nasal mucous membrane one finds a syndrome which is most prominent and well developed when the mucous membrane covering the cartilaginous portion of the nasal septum is a crimson red in color. This syndrome may be described as follows: These individuals are tired all the time. The day's work instead of being a joy represents a round of daily duties to be gone through with. They are irritable when they don't mean to be. They are a bit hard to live with when that is their least desire. They go about with a hair-trigger type of temperament and are easily thrown off balance by any unexpected situation arising during the day. While they participate in the three meals of the day there is no zest for food. In the middle of the day when they should be most awake they

are drowsy. When bedtime arrives and they should be drowsy they are most awake and often at their best. Their night's rest brings them only a moderate amount of refreshment, coming as a rule to the beginning of the day's work a bit tired. They are subject to indefinite symptoms of short duration in various parts of the body. They are subject to constipation. Their skin is dry and their hair comes out a bit more than they wish. They shun their social duties, feeling that the day's work represents the extent of their endurance. All in all, they feel a long vacation of some sort should be taken in order for them to feel right again.

The Red Septum Presyndrome: With the dark red mucous membrane covering the cartilaginous portion of the nasal septum there is associated a presyndrome, which may be described as follows: These individuals, while able to do the day's work, realize that they have very little reserve energy. They are able to follow fairly well a definite daily routine but experience difficulty in meeting a new situation which arises during the day's work. They find difficulty in surviving a short period of additional business or professional pressure or increased social duties. They prefer that their outside activities be mental, rather than physical. They readily fall a prey to the infections from which they usually recover. These infections are usually of an acute nature.

Interpretation of the Red Septum Syndrome: As to the factors underlying the production of the crimson septum and the syndrome, they seem to be three in number. In the order of their importance they are: First, crossing the line of tolerance for acid-ash producing food; second, long continued intake of medicinal acids; and third, fatigue due to excessive output of muscular energy. The trio usually met with consists of crossing the line of tolerance for acid-ash producing food, crimson mucous membrane covering the cartilaginous portion of the nasal septum and the syndrome. These individuals have a low tolerance for acid-ash producing foods, medicinal acids and an excessive output of muscular energy. Their metabolic process is apparently such that during metabolism a larger quantity of acids is formed than can be easily neutralized by the cell base, the intercellular fluids and blood plasma. In excreting this excess of acids resulting from metabolism, fixed base is removed from the body with a resulting negative base balance. The crimson redness of the mucous membrane covering the cartilaginous portion of the nasal septum enables one to suspect what is taking place in the body of the individual. The presence of the syndrome verifies this suspicion. The syndrome, in conjunction with the red septum, is most

helpful in deciding upon the type of treatment to be suggested in referred patients who are endeavoring to discover the reason for their loss of pep. In the office, the red septum and nasal discharge are most often seen together; while at the hospital, at the patient's bedside, the red septum alone often suggests the type of clinical attack to be recommended.

The Red Septum Treatment: When the nasal septum shows a marked increase in redness a number of requests are made of the patient. With reference to the first request, he is given a food list showing acid-ash producing foods in one column and alkaline-ash producing foods in another. Until his syndrome has disappeared and until the degree of redness of the mucous membrane covering the cartilaginous portion of the nasal septum shows a satisfactory decrease he is requested to select each meal three foods from the alkaline list to one food from the acid list. Following the disappearance of his syndrome he is requested to select one food from the alkaline list to each food selected from the acid list. In case a reappearance of the syndrome is noted, he is to return to the three to one ratio.

The second request is that he take an even teaspoonful of sodium bicarbonate in a glass of warm water at 10 a. m., 3 p. m. and 8 p. m. for three days. At the end of this three-day period he is requested to take a glass of lemonade, made from half a lemon without sugar, at 10 a. m., 3 p. m. and 8 p. m. for three days. Then sodium bicarbonate is taken again at 10 a. m., 3 p. m. and 8 p. m. for one day and lemonade again for three days. He is to continue this alkaline rotation of three days of lemonade and one day of sodium bicarbonate until one month has passed. At the end of the month he is to continue the lemonade once a day indefinitely.

The third request is to take one teaspoonful of cod liver oil at the morning and evening meal until he can sleep without morning fatigue. When this state of body is reached he is requested to continue the cod liver oil once a day indefinitely in order that a fairly large vitamin intake may be maintained. The degree of redness of the septal mucous membrane governs the dosage of the cod liver oil. If this mucous membrane is a fiery crimson in color, a teaspoonful of cod liver oil is prescribed at all three meals of the day. As the degree of septal redness diminishes the patient is requested to reduce the daily dose of cod liver oil until one teaspoonful once a day would represent the daily dose when the mucous membrane covering the cartilaginous portion of the nasal septum shows a dark red color.

The fourth request is to take a glass of tomato juice at one meal and a glass of some fruit juice at each of the other two meals. This

intake of tomato juice and fruit juices is to be continued at least one month and then modified as the patient wishes.

The fifth request is to drink a glass of water, plain or medicated, every hour from the time of awakening to the evening meal. This increased fluid intake is continued until puffiness of the eyelids or a slight bloating of the ankles is noticed. The time for this change to appear varies from a few days to weeks. When this state of body is reached the fluid intake is reduced to a glass every two or three hours during the day. The object of the increased fluid intake is to balance the intercellular fluids of the body.

Owing to the fact that I have been obliged to work alone I have not been able to develop the physiological side of the red septum as needs to be done. There are many questions within the province of the physiologist, the answering of which would be most helpful. These should be worked out if possible on the higher vertebrates, such as apes. Some of the questions are represented by the following: Why does the mucous membrane covering the cartilaginous portion of the nasal septum represent the only area that may serve as an index? Has this area a special nerve supply and we observe in this area the effect of sympathetic control of the blood vessels? Do blood vessels have chemical control as well as sympathetic control? If so, what part does each one play in bringing about a change in color of the mucous membrane covering the cartilaginous portion of the nasal septum? Why does a pale septum change to red much more rapidly than a red septum changes to pale under systemic treatment? Are results obtained by carrying the process of digestion farther or do we obtain results by unconsciously adding a catalytic agent in the form of minerals? Why may the degree of redness of the mucous membrane covering the cartilaginous portion of the nasal septum serve as an index as to whether incipient or actual staleness exists in the individual? Is it because the sympathetic control of blood vessels as observed in this area indicates the degree of tone of the nervous system as a whole? The answering of these and many other questions by the physiologist would be most helpful. Some of you are so situated that it is possible for you to work with a physiologist. I will greatly appreciate any co-operation you may give in trying to answer some of these questions.

Practical Value: By a study of the mucous membrane of the upper respiratory tract, especially that of the nose, you will be able to diagnose incipient or actual staleness in your patient. The presence of incipient or actual staleness in your patient serves as a handicap to securing your best operative results. It governs to some

degree the period of convalescence, whether it be of usual length or is drawn out over a longer period of time. By recognizing that the patient has crossed his line of tolerance for starches and sweets you will succeed in taking care of the residual nasal discharge and postnasal dripping which annoys the patient and allows him to conclude that your operative procedure has been only partially successful.

As a guide to preoperative or postoperative treatment the color of the mucous membrane covering the cartilaginous portion of the nasal septum assumes an added importance, for it points the way to acid or alkaline therapy. Apparently the blood vessels are susceptible to chemical control as well as sympathetic control. Under acid therapy they increase in size. Under alkaline therapy they decrease in size. The state of the tissues in the upper respiratory tract shares equal honors, it would seem, with the micro-organism in the production of clinical disease. Not being able to diminish the virulence of the micro-organism, we can often by acid or alkaline therapy render the body tissues unsuitable soil for the growth of this micro-organism. By a reduction in the starch intake we cause the mucopurulent discharge to disappear from the upper respiratory tract and by so doing remove a culture media for micro-organism growth.

Quarry Bank Building.

THE NASAL MUCOUS MEMBRANE AS AN INDICATOR OF FAULTY BODY CHEMISTRY. THE PALE SEPTUM.*

DR. IRA FRANK, Chicago.

Very little information concerned with the details of the physical and chemical processes in the body is available for daily use in practical ways in spite of the volume of investigation of human metabolism. This applies both to health and disease.

It has been stated that nutritional instability, by which is generally implied a disturbance of the acid-alkali equilibrium, is associated with many disease syndromes of a so-called constitutional type.

Patients present themselves without definite lesions but with general derangements, the cause of which it is not easy to determine. They may show a constant tendency to colds and other respiratory affections, or to infections elsewhere, or they may be irritable without cause, easily fatigued, subject to insomnia, to failure of appetite or to many other general symptoms. In examining these patients, the clinician collects a large amount of physical and chemical facts which he cannot ignore; but very often the real significant underlying fact is a nutritional disturbance depending upon an acid-alkali imbalance.

It is not my purpose in this paper to discuss the fundamental factors of nutrition and its disorders, except insofar as their clinical effects are concerned. I assume that an abnormal acid-alkali equilibrium does exist in nutritional disturbances, capable of producing characteristic symptoms. It may or may not be associated with other secondary factors.

In general practice there has been, so far as I know, no ready clinical method of discovering faulty acid-alkali balance in the body. The methods that we have of finding excessive systemic acid or alkali, such as urine and blood examinations, are tedious, time-consuming and require technical skill. It would be desirable to have a rapid, reliable clinical test, by which we could determine at a glance whether the nutritional state is faulty on the acid or alkaline side. Fortunately, such a sign seems to be available by inspection of the mucous membrane covering the cartilaginous nasal septum.

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Let us digress for a moment and consider the question of foods. The old classification of food into protein, carbohydrate, fat, minerals and water will not help us much in that chemical problem which deals directly with the acid-alkali balance. For our present purpose it is much better to consider foods as acid-ash producing and alkaline-ash producing.

The important pioneer work in this field for the past 14 or 15 years has been done by Dr. Jarvis, of Barre, Vt. The late Dr. Stuckey, of Kentucky, was interested in the early development of this problem. Dr. Jarvis has made careful clinical observations to determine the relationship between various types of nutritional states, and certain upper respiratory tract affections. His work has also been constructive by establishing the correlation between diet and respiratory tract symptoms. By changing the customary diet from an acid-ash producing to an alkaline-ash producing type, he believes that he has been able to favorably modify the symptoms. Dr. Jarvis' extensive and zealous pioneer work (and that of a number of loyal and enthusiastic colleagues who have accepted his views and are putting them into effect) is to be highly commended, and will, in my opinion, rank as a noteworthy contribution to medical progress.

In general, Jarvis has shown that proteins, fats, carbohydrates and mineral acids are acid-ash producing; vegetables and fruits, especially those ripened by the sun, are alkaline-ash producing. A more extended list of alkaline-ash and acid-ash producing foods is given as an appendix to this paper.

Various clinical manifestations may be produced on the one hand by an excess of acid-ash residue, and on the other hand by an excess of alkaline-ash residue.

The general effect of an excessive acid-ash producing diet and, incidentally, it may be remarked that the average American household dietary is excessively acid-ash producing, is what Jarvis calls a nutritional disaster; in fact, a chemical disaster. The nutritional disaster syndrome as described by Jarvis[1, 2] is as follows: These patients show continued lassitude, increased irritability, emotional instability, as well as anorexia. They show a reversal of the normal sleep habit, drowsy by day, wide awake at night; constipation, dry skin, easily fatigued and easily infected. *They show a crimson red mucous membrane covering the cartilaginous nasal septum.*

The individual with excessive alkaline-ash residue is usually spoken of as belonging to the allergic type; subject to colds, respiratory infections, hay fever and asthma. *The nasal septum mucosa is pallid and edematous.*

Glasscheib[3] states that vasomotor rhinitis is the expression of a blood alkalosis. Nasal transudates and exudates are common in such individuals. On the other hand, as Luscher[4] shows, in obstruction of the nose which prevents free respiration, a fall in the alkaline reserve of the blood results.

It may be remarked that the complete symptomatology in these types is not as yet definitely established, but the clinical sign that I wish to emphasize is the *color of the mucosa of the nasal septum.* A pale septum mucosa is generally a sign of alkalosis. The color of the mucosa of the nasal septum is, according to the foregoing, a ready indicator of the prevailing acid-alkali chemistry of the body. The degree of redness or pallor is governed by the composition of the food intake.

It is evident that variations in capillary permeability and dilation affect the quality and quantity of the blood brought to the tissues, a fact to which O'Keefe alludes especially in regard to proteins. But capillary instability may also be affected by food factors. Gänsslen has pointed to the red complexion of habitual meat eaters as compared with the pale visage of habitual vegetarians and states that the effect of a long continued meat (protein) diet is to dilate and enlarge the capillaries; on the other hand, a vegetable dietary tends to contract them. We may deduce that individuals consuming a continuous excessive protein diet, the nasal septum mucosa will be a bright red color, rather than a normal pinkish hue, due to dilation of the peripheral blood capillaries.

Any factor which modifies capillary circulation in general may be considered *prima facie* as altering the nasal mucosa circulation as well as its color. It is stated, for instance, that allergic stimulants dilate the peripheral capillaries also; indeed, O'Keefe thinks that the same physical stimulants that influence the capillary circulation are causal factors in allergic individuals. Walker mentions the case of a boy who whenever he took cow's milk, to which he was hypersensitive, showed vasomotor rhinitis, angioneurotic edema, asthma or other allergic phenomena.

It seems reasonable to conclude that the peripheral blood circulation is associated with the general type of diet and, consequently, with changes in the color of the nasal septum mucosa.

Returning again to the question of diet, as Dr. Jarvis puts it, will an individual receiving an excess of acid-ash producing food exhibit a bright red nasal septum mucosa, while one who is receiving an excess of alkaline-ash producing food exhibit a nasal septum mucosa which is pale? If the point of tolerance for starches is exceeded, a

mucopurulent discharge will be produced in the upper respiratory tract; similarly, if the point of tolerance for sweets is exceeded, the individual will generally show red turbinates with a stuffy nose and a watery discharge.

We believe then, that by the inspection of the nasal septum mucosa we derive valuable information concerning the individual's acid-alkali balance. In every instance a careful history should be obtained concerning the patient's food habits, to confirm and substantiate the rhinological examination.

From the foregoing the indications for treatment become apparent. In individuals in whom the symptoms are due to an excessive acid-ash diet, alkaline-ash foods should be substituted and suitable alkalies should be administered. For an individual whose alkaline-ash intake has been excessive, suitable dietary correction should be made. Many recommend the administration of dilute nitrohydrochloric acid before the dietary change. Jarvis prepares a solution of 5 drams of dilute nitrohydrochloric acid in water sufficient to make 4 ounces. One teaspoonful of the above dilution in a glass of water is given after each meal and at bedtime.

Beckman[5, 6] was one of the first to try the nitrohydrochloric acid treatment in hay fever. It has been mentioned and recommended by Gleason in his "Manual of Diseases of Ear, Nose and Throat" as early as 1914. Beckman had observed that the so-called allergic individual was one in whom there was at all times a slight disturbance of the acid-base equilibrium of the tissues in the direction of alkalosis. He compares the acid treatment of hay fever with the desensitization treatment, and finds that in 185 cases treated by acid, 40 per cent were completely relieved, and 26 per cent were markedly relieved; a total of 66 per cent successfully treated. In the desensitization treatment of 2,185 cases, 19 per cent were completely relieved, 51 per cent were markedly relieved; a total of 70 per cent successfully treated.

Beckman believes the allergic individual is one who is at all times in a state of a relative alkalosis. It should be added that Jarvis and a number of colleagues, who have become convinced of the soundness of his views in regard to the color of the nasal septum mucosa being an indicator of the acid or alkaline state, have reported a number of clinical cures following dietary correction, with or without added acid therapy. A prescribed diet which receives the patient's co-operation usually suffices, except in those cases of long standing, where permanent damage has resulted to the mucous membranes. Such a diet may be prescribed from the list of foods appended.

Of course, upper respiratory tract conditions with a pale or red nasal septum mucosa may be due to purely local conditions and in such cases local treatment alone usually suffices without systemic treatment.

Jarvis has reported a case of asthma in a boy with a pale edematous septum cured by acid therapy.

Lewis[7] considers that hydrops-mucosae, a common pathologic entity of importance in rhinology, in which the nasal septum mucous membrane is of a bluish pale gray color, is caused by a constitutional disturbance. He considers hydrops-mucosae in the nature of a chemical rather than an allergic derangement, which systemic therapeutics (acid) will probably alter.

He has observed that the symptoms and sensations of the patient in whom that syndrome occurs are those of upper respiratory tract irritations. These patients may also suffer from itching and burning skin, cracked epithelial areas about the auditory canals and mouth, associated spastic colon and spastic ureter, mucous colitis, arthritis, urticaria and edema. These symptoms yielded to systemic rather than to local treatment.

Personally, I have verified the importance of the nasal septum mucous membrane color sign in a number of cases, but I have found the pallid septum more frequent than the red. Probably this is due to the fact that the patient with the pale mucous membrane has a mucopurulent or watery discharge, which causes him discomfort and consequently he seeks relief by consulting a rhinologist. The patient with a crimson membrane is not uncomfortable and does not need to consult a rhinologist.

In closing, I desire to say that I have found these signs to be of value in diagnosis, and an important guide in treatment. I realize, however, that its ultimate value will be decided by a longer trial and by the final opinion of rhinologists.

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ACID-ASH FOODS.

These foods should always be balanced by adequate amounts of alkaline foods.

	Degree of acidity per 100 grams
Bread, white	7.1
Bread, whole wheat	7.3
Bacon	5.
Beef, porterhouse	10.9
Barley, pearl	10.4
Beef, ribs, lean	9.6
Corn, sweet, dried	5.95
Chicken	10.7
Crackers, soda	7.81
Cheese, cheddar	5.4
Corn, green	1.8
Eggs, whole	11.10
Eggs, white	5.24
Egg, yolk	26.69
Fish, haddock	16.07
Fish, pike	11.81
Fish, halibut	9.3
Fish, canned salmon	10.7
Fish, salmon trout	8.8
Fish, sardines	11.3
Fish, perch	6.3
Ham, smoked	9.7
Lentils	5.1
Mutton	9.6
Oatmeal	12.
Oysters, fresh	30.
Peanuts	3.9
Pork, lean	10.
Rice	8.1
Shredded wheat	12.2
Veal, loin	9.8
Walnuts	7.8
Cranberries	*
Prunes	*

*The ash of these foods is alkaline in nature but because of the unoxidizable acid contained by them they increase the acidity of the body.

ALKALINE-ASH FOODS.

These foods should always be used in adequate amounts to maintain a favorable alkaline balance of the body.

	Degree of alkalinity per 100 grams
Almonds	12.38
*Apples	3.76
Asparagus	.81
*Bananas	5.56
*Beans, dried	23.87
Beans, lima, dried	41.65
Beets	10.86
Beans, fresh string	5.4
Cabbage	4.34
*Cantaloupe	7.47
Carrots	10.82
Cauliflower	5.33
Celery	7.78
Chestnuts	7.42
Currants, dried	5.97
Cucumbers	7.9
Cocoanuts	7.
Dates	11.
Figs	10.
Grape juice	3.9
Lemons	5.45
Lettuce	7.37
Milk, whole	2.3
Molasses	56.
*Muskmelon	7.5
Mushrooms	4.
*Oranges	5.61
Onions	1.5
Peaches, fresh	5.04
Peas, dried	7.07
*Potatoes	7.19
Parsnips	11.9
Pineapple, fresh	6.8
Plums	6.2
Pears	3.6
Peas, fresh	1.3
Raisins	23.68
Radishes	2.87
Rutabagas	8.5
Sweet potatoes	6.7
Turnips	2.68
Tomatoes	5.6

*These foods have been found experimentally by Blatherwick to be the most efficient in reducing the acidity.

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BIOPHYSICS AND CHEMISTRY IN OTO-RHINOLOGY.*

DR. EUGENE R. LEWIS, Los Angeles.

At the request of the officers of this Society, I am presenting some general chemical and physical considerations, of fundamental and practical bearing upon many oto-rhinologic problems. While "considerations" are primarily limited to chemistry and physics, I have tried to extend them to include considerations of my hearers in this presentation; no attempt is made to go into details.

Medical theory and practice must be kept as closely as possible in accord with essentials of physiology and pathogenesis; concepts of pathogenesis, in turn, must conform to established tenets of biologic chemistry and physics; and the same rules must be observed in studying matter and energy in either living or nonliving form. In both forms, matter particles enter into chemical and physical combinations, attract or repel each other and undergo chemical and energy changes in exactly the same manner, and with the same general trend—*toward the attainment of a state of balance*—in movements, pressures, temperatures, densities, electric charges, hydrogen ion concentrations, saline, gas, fluid or other concentrations—toward an equalizing level called "equilibrium."

"Equilibrium" and Tissue-Reactions: In common with all combinations of matter and energy, those making up the body are constantly undergoing *equilibrium adjustments. Whatever takes place* within the body in the course of the infinite variety and uninterrupted succession of tissue changes (of fluid or colloid state, of chemical or electric potential, of absorption, imbititions, hydrogen ion concentrations and distributions of polar and nonpolar particles of all kinds), reflects *some* process in the course of *establishing* or *maintaining* equilibrium. Changing conditions in environment, chemical and physical trauma, emotional and psychic influences, in innumerable combinations, are endlessly calling forth such adjustments. These adjustments are *manifested* as "biologic phenomena," or "tissue-reactions"; but, primarily, physiologic or pathologic cell-potentials, functional activities or impairments, osmotic and circulatory pressures, tissue tensions, alkalosis or acidosis, colloid, saline and fluid changes all represent *tendencies of the body's energies and matter particles* to come to rest at the lowest possible energy-levels: in other words, to *reach a state of equilibrium;* only when cell-life *stops* do intracellular potentials — chemical and electrical — *reach*

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this state; *tendencies toward* equilibrium connote life, its *attainment*, death.

General Considerations: In this approach to the subject, physiologic phenomena (or pathologic variations) are looked upon as manifestations of the sum total of *reactions of tissues*. "Tissues" consist of body-*cells* and *what surrounds them;* "reactions" are the changes taking place in cells and their surroundings. In attempting to simplify this study, let us look at the cells, individually.

Cells are *composed of matter* and *manifest energy*. Unicellular organisms "live" very simply—by "reacting" with their natural environment; in this process these cells *take in* from their environment *energy-rich particles* and *throw off what remains* as "waste." These cells cease to live immediately the "environment" becomes unsuitable.

In a *polycellular* organism, such as man, each cell "lives" exactly as does the unicellular organism, *continuously "reacting" with "environment"* (contained in intercellular space), *continuously dependent* on environment for existence; after being appropriated from environment by the cell, *energies* are discharged in response to stimulation—during embryonic and immature life, as *cell-activities concerned with growth;* thereafter as cell-activities concerned *with function*.

The Cell an Engine: The cell is an *internal combustion* engine—its work is done by energy derived from fuel particles undergoing oxidation *within it*. Like all internal combustion engines, the cell depends upon the outside ("environment"—intercellular space) for constant *supply of fuel* and *removal of waste*, without either of which such an engine dies.

Environment: After entering the body as "food" and "breath," undergoing digestive and other preparations and traversing devious vascular and circulatory channels, *"environment"* ultimately comes into contact with the cell *in intercellular space*. "Reactions" taking place between environment (so projected) and the cell, constitute the actual episodes of "living." *Environment* thus appears of no less importance than the *cell*, as far as concerns *"reactions,"* or "metabolic changes," whose aggregates we recognize as *biologic phenomena*.

Cell-Properties and Cell-Nutrition: The properties of the cell are determined by the *properties of its component matter-particles** and

**Nonliving matter-particles*, on contact with the cell, are taken in through the cell wall, whereupon they take part in living processes—they are *actually incorporated into the material composing the cell*. The precision with which free-moving particles arrange themselves in conformity with cellular structural pattern resembles the behavior of constituent particles according to structural pattern of the crystal.

energies entering into its makeup. The cell is *unique*, in that it embodies an agency by virtue of which successive particles, as they are taken into the cell, arrange themselves according to its own peculiar *structural pattern*. The structural pattern of each individual cell differs from that of any other cell; yet each cell is able to continue to live, even though considerable physical and chemical change take place in its surroundings; it can "adapt" to a wide variety of "fuel" or "food." Thus, alterations in the *actual chemical constitution of the cell* are constantly taking place, as chemical alterations in food and breath effect continuous changes in the *chemical composition of the contents of intercellular space*.

Physics of Cell Nutrition: 1. It is by no mere chance that particles enter or leave the cell; *forces* are required to move them. *At all times* both forces and matter-particles are governed by laws of physics and chemistry, in accord with which free-moving particles come to be *selected* from outside the cell, *pass through* the cell wall, become *component parts* of the cell, *occupy (momentary) positions* with relation to other component particles in the structural pattern, and *undergo changes in chemical identity and energy-content* as they *participate in cell activities* before being *thrown off as waste*.

2. A red blood cell immersed in water swells, and ultimately bursts; but if normal environment is restored early enough, these cell changes undergo reversal and the cell resumes its former condition. "Swelling" results as the cell absorbs water from the outside; reversing conditions, intercellular space absorbs water from the cell.

Reviewing briefly: *Body tissues are composed of (1) cells,* in contact with (2) intercellular matter-particles and fluids, *i. e.*, "environment." *Reactions* between *cell* and *environment* convert (into *living state*) *incoming particles* of *energy-rich matter;* stimulus *discharges energies* of these incorporated particles as *cell activities* (of *growth* or of *function*); what remains is thrown off as *cell-waste*. Under various electrical and catalytic (nervous and endocrine) influences, the manifestations of *all* tissue-reactions taking place in the body at any time are seen as "symptoms" and physical signs." Reactions between cell and environment following certain chemical changes may result in *cell-death*, as from carbon monoxid particles; or in profound *alterations* in the *form, size* and *condition* of the cell.*

Biochemical Aspects of Pathogenesis: Chemicals *inimical* to cell life are termed "toxins"; the *toxicity* of a substance is determined by

*During asphyxiation, cell death impends; if oxygen is restored in time, the *cell recovers;* heat, electricity and nerve *forces* alter tissue-reactions, with resulting *death* or *recovery* of the cell, as the case may be.

the outcome of its reactions with the cell. A cell may become *abnormally susceptible* to toxins (for example, in "sensitizations" to antigens), or *abnormally immune* (for example, morphinism), depending upon its *chemical constitution of the moment;* at different times the *same cell* may show tremendous differences in its reactions to the *same substance.* A cell may thrive on certain tissues at one time, very poorly or not at all at another time.

Coupling up with consideration of infectious troubles and *sinus disease,* this brief review of cells, cell-environment and reactions is presented as a preface to considerations of the clinical signs and symptoms of the tissue-reactions which constitute the status of "health" and "disease." What has been mentioned concerning the cell applies to bacterial as well as to body cells.

Chemical Aspects of Infection and Convalescence: During the early stages of infectious disease, the patient's tissues are *"favorable" to growth* of bacterial cells; during convalescence the same tissues are *"unfavorable."* This difference reflects changes in *body tissues,* rather than in bacteria, for those from a convalecsent reproduce disease in the tissues of a new host.

Pathogenic *bacteria universally* exist in contact with healthy body tissues without any evidence of their presence; following some profound disturbance—shock, fatigue, exposure or traumatism—*pathologic changes and symptoms* reveal the *beginnings of "infectious inflammation"*; and biopsy or bacteriologic examinations reveal *infection* by the very streptococci, pneumococci, etc., whose presence had *hitherto failed* to produce such tissue-changes. The momentary condition, or constitution of body tissues ("favorable" or "unfavorable" to cell growth of bacteria), determines the actual onset of most *acute infectious diseases.* Chemical and physical changes taking place in the body convert tissues from previously "unfavorable" into "favorable" media for bacterial cell-growth; and the onset of convalescence signalizes reverse changes in tissue-conditions. Chemical changes in tissues are determining factors in susceptibility to, and recovery from, infections.

Effects on the Cell of Changes in Intercellular Space: The contents of intercellular space are changed *chemically* by whatever enters the circulation; the chemistry of intercellular particles and fluids determines to a considerable extent the *momentary chemical constitution* of body cells. As already stated, the *properties* of the cell are influenced by the properties of its constituent particles; *cell-reactions vary,* as changes take place in *chemical composition,* either of *cell* or of *cell-environment.*

Physical Changes in Tissues: Change in chemistry of cell or intercellular content leads to *physical changes* in tissues—alterations in size, shape, temperature, texture and distribution of fluids. Among the first *physical manifestations* of altering chemistry is local interstitial *hydrops* or *dessication*, depending upon *changing osmotic pressures* in the cell and intercellular space. In the process of *restoring osmotic equilibrium* the cell may absorb abnormal quantities of fluid from intercellular space; or *vice versa*, abnormal quantities of fluid may be absorbed from the cell by the contents of intercellular space. Clinical picture of *such dessication* is seen in *the parched, red*, friable mucosa; *of hydrops* in the soggy, pallid, polypoid mucosa. Secondary physical changes in tissue are often superimposed upon these. As bacteria permeate tissues they provoke *two additional kinds of disturbance*—by their *presence*, "reactions to foreign bodies"; by their *cell-waste*, "reactions to bacterial toxins." The symptoms and physical signs elicited on examining a patient are *bodily manifestations of tissue-changes* whose physiochemical nature has been reviewed briefly. It is beyond the scope of this paper to point to their specific bearing upon oto-rhinologic problems; in no less measure, they bear upon other similar medical problems—spasmophilia, abnormal fluid metabolism, so-called "sensitivities"; gastric ulcer and colitis; pyelitis and cystitis; neoplasia; catarrhal endometritis or vaginitis — leukorrhea; ocular hypertension; hyperceruminia; urticaria and eczema, epilepsy, migraine and chorea; arthritis; abnormal circulatory tensions. In different patients, or in the same patient at different times, diagnosis depends upon the "chief complaint"; the patient may complain of sinus trouble, renal colic, pruritis, colitis, leukorrhea, epilepsy, etc. From the standpoint of body-chemistry, *primary etiology* is much the same in all such conditions; the terms used denote differences in *the site* and the *kind of tissue* manifesting local effects of such underlying etiologic factors.

The consequences of increasing differences of rhinologic opinion concerning the etiology and rational treatment of nasal sinus troubles are beginning to include troubles of another kind. Most *referred patients* are expecting *local treatments only*—antral, ethmoid or turbinate operations, sinus lavage, or whatever their doctor may have mentioned; before coming to the rhinologist these patients have usually discussed such matters more or less in detail with the family physician, whose general impression is that the troubles, which *he does not want his patient to have*, are "being removed" daily, by suitable rhinologic procedures; and current literature continues to

justify his impressions. *Many physicians* have also come to expect the rhinologist to *confine himself to local treatments,* and are inclined to resent another's suggestions as to *diet* or *other general treatment* for their patients.

Because those exclusively concerned with such problems hold such widely divergent opinions as to "sinus troubles" and their treatment —one group convinced of the efficacy of operative removals (of mucosa and other tissues found to be diseased), another group equally sure that most *sinus troubles* are neither so "removed" or "so removable"—the rhinologist owes it to his patients, to his profession and to himself to *undertake the settlement* of certain general questions bearing directly upon the *nature* of nasal sinus troubles and their *causes.* The determination of certain facts, not difficult to prove, will go far to dispel *many misconceptions as to the nature* of sinus troubles, and *many differences of opinion* regarding the two fundamental essentials—*cause and treatment.*

For many years increasing experiences with my own patients, as well as with those of others, have convinced me that *clinical results alone* more than suffice to disprove localistic concepts as to pathogenesis: careful testimony as to the *ultimate* effects upon a large majority of patients *fails to show* that persistent local treatment of any kind, with or without radiations or removals of nasal and sinus tissues, *has ended their troubles;* indeed, in a strikingly large number of cases, complaints *have very definitely increased* following such procedures. Three years ago, and again last winter, I presented to the American Laryngological, Rhinological and Otological Society accurate analyses of over 1,600 consecutive cases of "sinusitis." Histories, metabolic rates, tissue tensions, circulatory pressures, weight variations and hemograms show clearly that most sinus troubles are only *part of a larger problem;* complete studies show various *systemic* and *other local manifestations* of some underlying cause, *without elimination of which* recurrences of its local manifestations are inevitable — regardless of transitory ameliorations following many kinds of local treatment.

General systemic measures supplementing any kind of local treatments of upper respiratory disease are determined, *primarily,* by *considerations of factors in pathogenesis; secondarily,* by *clinical progress* checked by adequate supervision. Oto-rhinologic experience and training should offer prospect of most valuable judgment —both in evaluating the significance of all findings and in deciding upon, and applying, general and local therapeutic measures best suited to meet the indications.

The patients whose sinus troubles are *caused by local nasal* pathology is readily identified *by the absence* of significant findings on examination, particularly the family and personal histories, the blood picture, basal metabolic rate, blood and pulse pressures, body weight and general physical conditions. He is the *unusual* patient. The great majority of patients with sinus troubles show unmistakable evidences of underlying etiologic abnormalities, which will continue to be revealed in the form of occasional or frequent attacks of "sinus-troubles" *as long as the underlying causal conditions* continue to exist.

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A worth while article on "Tracheotomy—Its Indications and Management," by L. C. McHenry, appears in the *Journal of the Oklahoma State Medical Association* for September, 1932. The author cites low tracheotomy as the only modern technique with the two exceptions of emergency and prelaryngectomy when a high tracheotomy may be necessary or advisable. Cases of stenosis of the larynx due to improper technique bear witness to the soundness of this dictum. H. C. R.

Jose R. Perez, in the *Journal of the Philippine Islands Medical Society,* August, 1932, reports a series of acute and chronic otitis media purulenta treated by intramuscular injections of sterilized milk. While his records of the cases are inadequate, his results are so startling that they are deemed worthy of mention. He treated seventy-two cases in all, ranging in age from one month to 18 years. Sixty of his seventy-two cases were completely cured; ten cases improved, and two showed no improvement. It was later demonstrated that these two were cases of tuberculous otitis media. He described three cases, two of which were distinctly chronic otitis media and both of which cleared up after relatively few injections.

HEAD INJURIES AND EAR DISTURBANCES.

Dr. A. J. Guerinot, Pittsburgh.

Connections between head injuries and the ear. The brain is affected first and then the ear.

There are three groups: *1.* From concussion of brain with ear symptoms. *2.* Concussion of brain with concussion of the inner ear *3.* Concussion of brain with fracture of temporal bone.

1. Concussion of Brain: Bones of the skull compressed after injury. If not severe, bone goes back, or, if it does not, you get fracture. Skull compression gives acute increase of brain pressure.

Contents of Skull: Brain substance, blood vessels and cerebrospinal fluid, excluding meninges.

The brain substance will be compressed. This is called substance compression of the brain.

Blood vessels (Ricker): If injury touches blood vessels, you get a praestatic blood circulation, which is functionally characterized by the fact that the blood runs slowly in a large blood vessel. (In inflammation, blood vessels are enlarged but the blood runs quickly.) Anatomically, the praestatic circulation is characterized by the fact that exudate and lymphocytes go out of the blood vessels, as in chronic inflammation. This may be a persistent condition.

Fluid: The lateral ventricle will be compressed through the foramen Munroe into the third ventricle. From the third ventricle goes into the aqueduct of Sylvius. From the aqueduct of Sylvius to the fourth ventricle. Cannot all go out because the spinal cord canal is too small, hence fluid pushes the walls of the fourth ventricle, especially the floor. In the floor of the fourth ventricle are the nuclei of the vestibular and the cochlear nerves. After concussion of brain the findings in brain substance depend upon the length of time. If the patient dies, say in an hour, you cannot find anything. Every concussion produces changes in the brain texture, which are degenerative changes, which can be localized in various places of the brain, as the striate body, the optic thalamus, cochlea and vestibular nuclei. Found in 42 per cent of head injury cases, cochlear and vestibular nuclei. These changes appear clinically in the form of concussion of brain with ear symptoms. Nothing is found in the peripheral ear.

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Symptoms: *1.* No tinnitus. *2.* Hearing normal. Bone conduction normal also. If patient hears, in bone conduction, six seconds shorter than examiner this is called bone conduction shortened. *3.* Dizziness found often. *a.* Characterized by the fact that it is not intensive. If he has much dizziness, he probably has bleeding in the posterior fossa. *b.* Characterized by the fact that it comes in attacks. If it is persistent, it is more than a concussion of brain. *4.* Disturbance in equilibrium. Either not present at all or just small in amount. If marked, has something more than concussion. *5.* Nystagmus. As a rule, weak; not always present. If nystagmus is intensive it is probably bleeding. *6.* Pointing test. Without importance. Head movements, from time to time, positive. Caloric tests, normal or hyperirritability.

Prognosis: Concerning dizziness, very bad. Say it will be persistent, or increased. How can you prove dizziness? If you have intensive nystagmus, the patient has dizziness. There are two methods: *1.* Positive head movement test. *2.* Labyrinthian hyperirritability, or both together, then you are justified in saying he has dizziness. If the patient has spontaneous nystagmus, he has dizziness and you do not have to examine further.

Loss of 40-60 per cent ability to work. Reasons: *1.* Cannot work as before, as on roof. *2.* Prognosis is bad. *3.* No treatment. Can only give good advice, bromides, hygiene, etc.

2. Concussion of brain with concussion of inner ear. In what way does the concussion of the brain affect the inner ear?

(Theory) By blood circulation. Internal auditory artery is chief artery of the inner ear. Comes from the basilar or cerebellar artery. You can get praestatic circulation in the inner ear. This produces what is called otitis interna vasomotoria—exudate and lymphocytes around blood vessels. (Also found in climateric; after poisoning; also tertiary stage of lues.) In the latter cases may also be in the posterior fossa. May give typical cereballar symptoms, called vessel crises in posterior fossa.

Diagnosis of otitis interna vasomotoria: *1.* Hearing always disturbed, most often on one side. *2.* No tinnitus. *3.* Spontaneous labyrinth symptoms of high degree. *4.* Labyrinthian irritability is, as a rule, changed.

The caloric test is either normal, or less than normal. Never more than normal. May be completely absent.

Turning normal in the majority; absent in a few.

Prognosis is bad concerning dizziness and hearing; 40-60 per cent loss. Deafness on one side is not an obstacle; 10 per cent loss.

Treatment: Calcium and iodin. Iodin-sajodin tablets; three to four daily, or "Iodipin," 100 c.c., add five drops of oil of peppermint, of mixture give three drachms daily. Calcium lactate not good, because it does not work.

"Afenil" calcium urate in ampoules, give once a week. Before using: *1.* Heart and blood vessels must be normal. *2.* Intravenous, absolutely. *3.* Slowly, otherwise get collapse. If it works, you get a red face. Prickling at tip on tongue.

"Atrocal" tablets; three kinds: *1.* Children; *2.* adults; *3.* strong atrocal. Adult's atrocal, four to six tablets daily. This also works in cases of otitis interna vasomotoria; of climateric. They have dizziness and tinnitus.

3. Concussion of brain with fracture of the temporal bone: *1.* Longitudinal. *2.* Cross-fracture. *3.* Rupture of tip of petrous bone. Die quickly.

1. Longitudinal fracture is one which starts most often in tegmen and goes through the tegmen to the roof of the Eustachian tube. As a rule, linear, no branches. If branches, they go either into mastoid process or bony external canal, but not into the inner ear. (Microscopic fracture in inner ear only found in Paget's disease.)

Typical middle ear fracture: Changes produced: *a.* Drum ruptured. *b.* Middle ear either compressed or partially filled with blood. *c.* Middle ear muscles, especially tensor tympani, will be destroyed. *d.* Ossicle chain will be in the majority of cases not touched. In the minority there is a subluxation of incus. *e.* Inner ear. Hemorrhage petechial, found only in cases that die immediately. In cases that survive nothing is found or else otitis interna vasomotoria. Healing: Does not heal with bone, but with connective tissue.

Diagnosis of longitudinal fracture: *a.* Outflow of blood from external canal does not prove it even if arterial. If the cerebrospinal fluid comes out then you have fracture and rupture of the dura. Patient looks terrible. In severe cases where fluid comes out in great amount they may heal if let alone. Only danger is in treating them, when they may get meningitis. *b.* Findings in external canal. Hyperemia in posterior superior quadrant does not mean much. Fracture here in superior wall; you can be sure he has fracture in middle ear.

In recent cases of injury to head do not examine ears because of danger.

Changes in drum: Hematotympanum. You can see blood through drum; drum looks black.

Functional tests: 30 per cent of cases have diminished hearing.

X-ray: Reliable if positive. If negative do not exclude longitudinal fracture because it may be very fine. Longitudinal fracture very common in head injury. Better to diagnose it than to exclude it.

2. Cross-fracture: Starts in the region of jugular bulb, crosses the pyramid and ends on the anterior surface of the petrous bone. Typical inner ear fracture destroys inner ear; vestibulum. Cochlea and semicircular canals as a rule only destroyed by the bleeding. Clinically important—does it touch the lateral wall of canal or inner window? If the windows are destroyed there is a pathway for infection from throat via the Eustachian tube to the brain.

Healing: Does not heal.

Diagnosis of cross-fracture: *1.* X-ray: More reliable than in longitudinal because they are more open. *2.* Functional tests. Deafness. No irritability. *3.* Drum picture. If drum is ruptured it is probably only longitudinal fracture and, therefore, hearing is better. If drum is not ruptured it is a cross-fracture and hearing is bad.

Prognosis: *1.* Life. *2.* Function.

1. Life. Life in longitudinal fracture questionable. Do not forget that fracture does not heal. May get a meningitis. Seven per cent of cases have meningitis.

2. Function. Prognosis concerning hearing not good.

1. Life; in cross-fracture it is questionable. Ten per cent of cases have meningitis; don't heal. Function hopeless.

Treatment: Partly conservative and partly surgical. Sterile gauze in ear. Don't examine. Don't remove blood. Cold to ear.

Surgical treatment: Primary trephine, 24 hours after injury, because a big part die of increased pressure and only 8 per cent die of meningitis. Primary trephine indicated if you find in spite of spinal puncture 24 hours after injury signs of increased brain pressure. Signs: Unconsciousness, mydriasis on sick side. Changes in cerebrospinal fluid, chemical.

Contra-indications: *1.* Moribund patients. *2.* In patient with many fractures or ruptures. *3.* Children. *4.* With clear interval diagnosis: tuberculosis, lues, severe arteriosclerosis. Trephine where fracture is located. If no signs are present do a subtemporal decompressing of Cushing.

Treatment of head injury combined with running ears.

Group 1: Cases of mucous membrane suppuration which are combined with head injury, usually recover without operation. Wait until you get usual indications for operation, as mastoid symptoms, fever, etc.

Group 2: Chronic bone suppuration, cholesteatoma and injury. Absolute indication for operation. (Pregnancy influences ear condition very much. This changes a relative indication to absolute, especially in ear.)

Group 3: Acute otitis after head injury. Paracentesis. If patient gets meningeal symptoms, operate. If you operate, operate completely; radical, if necessary. Expose whole fracture. If it goes into middle ear, do a radical operation.

604 Westinghouse Building.

SPONTANEOUS RUPTURE OF THE SIGMOID PORTION OF THE LATERAL SINUS.

DR. JAMES A. FLYNN, Washington, D. C.

Spontaneous rupture of the sigmoid portion of the lateral sinus following mastoid surgery is of such rare occurrence that the following case report should be of interest:

Case Report: B. M., age 9 years, was first seen by me, March 31, 1932, in consultation with Dr. John Harrington. Her chief complaint was pain and tenderness in the mastoid region.

Family History: Irrelevant. *Previous History:* When 14 months of age she was sick with an intestinal infection caused by milk. Mother reports that the child was very ill, with high fever and delirium. Head colds and some cough have been present more or less during the child's entire life. At 3 years of age, when living in Oklahoma, a tonsil operation was done; however, it was necessary to repeat this operation in 1931. During her sixth year she suffered an attack of chickenpox. In 1928, she was inoculated against diphtheria. Measles developed early in 1931, followed by an attack of acute otitis media, developing into mastoiditis. The mastoid disease was treated surgically. Four weeks following this mastoid operation it was necessary to return the patient to the hospital and again open the wound. After a protracted convalescence the child was apparently well.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 9, 1932.

Present Condition: A cold of several days' duration, accompanied by a productive cough. No other symptoms. On March 21, 1932, she complained of pain in her right mastoid, which became markedly tender on palpation. There is no discharge present, but the membrana tympani is deeply congested. Temperature at 5 p. m., 102°; pulse, 130.

Physical Examination: Heart and lungs essentially normal; marked tenderness over mastoid area; considerable swelling of entire mastoid area; deep depressed scar resulting from previous operation; urine normal. Blood examination: Hemoglobin, 73 per cent; red cells, 3,680,000; white cells, 28,600; differential count resulted as follows: lymphocytes, small, 21; lymphocytes, large, 6; polymorphonuclear, 73 per cent.

Diagnosis: Acute recurrent mastoiditis.

Operation: March 22, 1932. Postauricular incision was made around the scar of previous operations. The tissue was easily separated from the bone. A small excavation in the mastoid bone was found, but the antrum had not been opened. The bone was then removed, the antrum found, and a probe passed through the aditus; the dissection was continued toward the tip, then posteriorly, when rather soft, definitely diseased bone was encountered. This bone was removed and the sigmoid was exposed from the knee down for about one-and-a-half inch. Superiorly and anteriorly, the soft bone followed into the zygoma and the dura was exposed. The sigmoid was palpated, it was soft and resilient; but the walls of the sinus appeared thickened and of an unusual white color. A diagnosis of periphlebitis was made, but as no chill had occurred and no complaint of earache nor other localizing symptoms, I decided to pack the cavity and await developments. The cavity was closed with three skin sutures and the patient returned to bed. Temperature, 101°; pulse, 122. The remainder of the day the patient slept and had a good night following.

March 23, 1932: Good day and night.

March 24: Patient felt good, ate soft food, had a good night, but temperature went to 104° at 4 p. m. Wound was redressed and dressing was filled with pus.

March 26: Patient comfortable and very cheerful, ate well and slept well; temperature down to 99.5°; pulse, 100; respiration, 20; blood count this day: leukocytes, 16,300; lymphocytes, small, 24; Large, 4; polymorphonuclears, 71 per cent; mast cell, 1 per cent.

March 27: Very good day and night, temperature normal, pulse, 100; respiration, 120. Patient redressed and after removing the

packing a sudden rupture of the sinus, close to the knee, occurred. This was stopped by pressure and the child removed to operating room to complete the dressing. The rupture was large and it was found that bleeding occurred from both ends and no thrombus was present. Iodoform gauze rolls were inserted into the vessel to stop the hemorrhage.

March 28 to 31: Very uneventful, temperature remaining normal until the afternoon of March 31, when it reached 102°; pulse remained 90 and 100 during these few days. Patient very comfortable, ate well and slept normally.

April 1 to April 7: Temperature ranged between 99° and 103°. Blood count, April 2: Hemoglobin, 73 per cent; red cells, 3,680,000; white cells, 28,600. Lymphocytes: small, 21; large, 6. Polymorphonuclears, 73 per cent. A transfusion of 300 c.c. citrated blood was given, April 7. The temperature continued its irregular course, ranging between 99° and 103° until April 10, when it came down and remained normal. Another transfusion was administered, April 10, of 300 c.c. citrated blood. The object in giving the blood was to combat the infection which was evidently present and to increase the coagulability of the blood.

A definite paresis of the right side of the face developed on April 5, the mouth was drawn to the left side, the muscles were flabby, and the eye of the affected side could not be closed. This condition continued over the following day, April 6, and then disappeared as suddenly as it came. My conclusion is that it was due to pressure from packing in the mastoid.

Cases reported of spontaneous rupture of the sigmoid sinus are rare. I have been able to find but two in the literature of recent years. Williams reports a spontaneous rupture of the lateral sinus following mastoidectomy in a child of 5 years. Seven days following operation, rupture with profuse hemorrhage took place. It was controlled by packing. Williams states that the sinus was not thrombosed.

Hill reports a case of spontaneous hemorrhage four days after simple mastoid operation. The patient developed septicemia with metastatic manifestations. Jugular ligation and treatment produced a complete recovery.

Comments: Why have three operations been performed on the patient's mastoid? The first reason is that the two operations done after the primary infection were incomplete. The antral cell had not been opened nor had the bone dissection gone far enough to remove all diseased bone from around the sinus.

The presence of this diseased bone undoubtedly spread to the sinus, causing a periphlebitis. The removal of the diseased area a year later exposed a diseased vessel that could not stand the normal pressure within. How can we remove all diseased bone during the performance of a mastoid operation? My answer is to have a systematic method of procedure. Portmann visualizes the mastoid into definite areas as follows: Antral portion deep and superficial deep antral gutter leading down to the tip cells. Zygomatic area. Area of the sinus deep and superficial. After the bone dissection is complete use 10 per cent zinc chlorid to differentiate healthy from diseased bone. Healthy bone is white, diseased bone dark.

In a recent examination of my patient I find that both maxillary antra are clouded and a very definite left side ethmoiditis is present. We can now account for her almost continuous head cold, which was referred to in her history. The two tonsil operations reported evidently did not remove the source of infection. Had a careful examination been made after her first operation the ethmoiditis with polypoid middle turbinate area would have been discovered. Therefore, my criticism would be to make a most careful examination of all sinuses, the pharynx and tonsils in all children complaining of frequent head colds.

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1511 Rhode Island Avenue, N.W.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of May 13, 1932.

Dr. Douglas Wood, President, presided.

The following cases were presented:

Dr. G. M. Constans reported five retropharyngeal abscess cases as follows:

Case 1 (No. 39453A): A. G., male, age 40 years, laborer, was seen on March 9, 1929. Ten days prior to this time he had sore throat, accompanied by inability to eat, dyspnea and high temperature. He was sent down by his local physician, and on the way down the mass in his throat ruptured, discharging pus. After this he felt better.

Examination showed the pharynx to be injected, tonsils of moderate size, and a sinus in the retropharyngeal space above the right arytenoid region which was discharging pus. There was some swelling of the right neck. Otherwise the examination was negative.

Diagnosis: Spontaneous rupture of retropharyngeal abscess.

Case 2 (No. 50210): R. F., male, age 35 years, advertising man, was seen on Sept. 17, 1930. He stated that two days prior to being seen he had trouble with swallowing, particularly in the left side of the throat. This was accompanied with pain and general malaise.

Examination showed congestion of the throat. The following day a small retropharyngeal abscess was noted on the left side of the throat. There was also some swelling of the epiglottis.

The abscess was incised and the patient made an uneventful recovery.

Case 3 (No. 977): F. B., male, age 64 years, hotel proprietor, was seen Dec. 24, 1931. This man was brought in by his home physician, and stated that he had been ill for three weeks with soreness and pain in the throat, difficulty in swallowing, and dyspnea.

Examination showed the throat to be injected, submerged tonsils, and slight glandular enlargement of the neck. There was a small mass in the retropharyngeal space, but this was not very definite. The following day the mass attained considerable size and was definitely palpable, so it was incised and drained, and the patient gained rapidly following this procedure. The abscess ceased draining in about four days.

Case 4 (No. 13265): M. H., male, age 6 months, infant, was seen on Jan. 9, 1932. The baby had been seen on the previous day by a pediatrician at home, with a history of having been ill for two days. There was great difficulty in swallowing, mucous in the throat, difficult breathing, and high temperature.

The patient was sent to the hospital and examination showed moderate injection of the pharynx and nasopharynx. There was no obstruction in the tonsil region but obstruction was felt down lower, where a rounded mass was made out on palpation.

Diagnosis: Retropharyngeal abscess.

X-ray examination showed the trachea to be markedly displaced forward, and the X-ray diagnosis was retropharyngeal abscess.

Incision and drainage was instituted and pus obtained. This continued to drain for a period of nearly a week. The child then developed a bilateral otitis media, from which he subsequently recovered. (The child has also had pneumonia since that time.) X-ray taken five days following incision showed the trachea back to normal position and the chest clear.

Conclusion: Retropharyngeal abscess; healing.

Case 5 (No. 51454): D. H., male, age 5 months, infant, was seen by the writer on March 5, 1932. This baby was referred by the pediatrician who first saw the child for the present illness about Feb. 22, 1932. At that time there was considerable coughing with dyspnea. The patient was running a temperature and seemed quite ill.

The baby was admitted to the hospital, March 4, 1932, with a diagnosis of questionable retropharyngeal abscess and bronchopneumonia. By palpation a very slight swelling was made out in the retropharyngeal space but nothing really definite. X-ray examination showed a retropharyngeal abscess and resolving infection in the right lung. Under local treatment the child steadily improved. A re-ray was taken on March 8, 1932, and no change over the previous examination was noted. On March 9, 1932, further investigation was made, but no abscess mass could be felt. The region was aspirated with a needle but no pus obtained. In this case, however, the patient went on to an uneventful recovery. Apparently the abscess either absorbed or ruptured through a small opening which was not made out.

Diagnosis: Retropharyngeal abscess.

DISCUSSION.

DR. CONSTANS stated there was one point to be made in these cases, and that is they are not very uncommon and in infants, particularly, he thought the X-ray was especially valuable in diagnosis in showing the displacement of the trachea.

DR. E. A. LOOMIS said he wished to mention one experience he had had before he began specializing. This was with a general practitioner who had called Dr. Loomis to help him hold a child and told him he was going to open a retropharyngeal abscess. This doctor said in a case of this kind when one made a cut he should make a good big one. In this particular case, it happened to be a misplaced internal carotid that he cut, and the child passed out right in the doctor's office. Dr. Loomis said since that experience he himself had always aspirated these cases to find out if there is pus. Then if one finds pus he can operate safely.

DR. J. H. GAMMELL said he had seen two such cases with pediatricians. The history seems to follow very much the same course as the development of a quinsy; there may be a tonsillar sore throat apparently getting along nicely and then the patient begins to have difficulty in swallowing and breathing. Dr. Gammell recalled one interesting case of a youngster between two and three years of age that went along nicely for about ten days and then began to have trouble; breathing was bad and there was considerable phlegm. One side was opened. The patient seemed to be getting along nicely for a while and then about four days later Dr. Gammell was called again, when he found another abscess on the other side. In one family a girl about 8 years of age had a retropharyngeal abscess, and the next winter after this patient had the abscess the younger sister, about two years younger, had the same thing. Dr. Gammel said he did not know whether it runs in certain families, but there were two in this particular family.

DR. W. E. CAMP stated that one sees a good many of these cases in children, but not so many in adults. He felt that one of the things which makes one too optimistic is seeing them early. One of the complications is mediastinitis, where the pus burrows down into the mediastinum. Dr. Camp had had one of these cases that had gone on for some time. It was decided to open it without an anesthetic, so the child was mummified in a blanket and a mouth gag was inserted. The child suddenly died. An autopsy was obtained and it was found that the pus had gone down into the mediastinum so this child would probably have died anyway. Dr. Camp felt that this goes to show that early diagnosis of these cases is very important.

DR. CONSTANS in closing, said, along the line of treatment, it is fairly obvious that these patients should be placed with the head well down and precautions taken so they will not aspirate any pus. He does not use the mouth gag. He thought Dr. Loomis' point was well taken; he himself aspirates these cases first and then operates, thus giving one a little idea of what he is working with.

Malignant Tumor of the Nasopharynx with Involvement of the Nervous System. Dr. Harold J. Rothschild.

The patient, J. M., Chinese, sculleryman, age 41 years, was first seen by Dr. Rothschild on Dec. 9, 1931, complaining of the following symptoms: (1) Diplopia; (2) right-sided earache and moderate deafness; (3) numbness of the right side of the face; (4) a feeling of obstruction in the back of the nose and throat, and blood-tinged discharge.

The history in the case was that the diplopia began suddenly one year previously without preceding illness. The ache in the right ear began one year previously and was followed a short time later by moderate loss of hearing. The numbness of the right side of the face began one month previously and was preceded by an itching sensation of the same area for four months.

The discomfort in the back part of the nose and throat compelled him to make efforts to clear them, and streaks of blood had been noted in the mucoid secretions expectorated for the past five years.

He had recently received thorough examinations elsewhere, including neurological, spinal fluid, eye, ear, nose and throat, X-ray of the head, and no diagnosis had been forthcoming.

The notes of the writer's examination of this patient are as follows:

Eyes: A left abducens paresis was found to account for the diplopia, which has since disappeared. The pupils were equal in size and reacted equally and normally to light and accommodation. The visual acuity in the right eye was 20/20, left eye 20/25. The refractive media and fundi were normal. Visual fields were full for form and color.

Ears: The left ear was normal. The right ear revealed a retracted drum with the appearance of mucoid secretion in the middle ear. Tuning forks revealed conduction type of deafness. Whisper could be heard at four inches, increased to 1½ feet after inflation.

Nose and Throat: Negative, except for a mottled bloody appearance of the pharyngeal vault with some mucopus bathing this area, especially to be noted on the right side. Holme's nasopharyngoscope revealed a narrowing of the nasopharynx and hemorrhagic blebs and bloody streaks on the posterior portion of the right nasal mucosa extending into the pharynx. The lips of the right Eustachian orifice were swollen and injected.

The patient was referred to Dr. Ruhberg, neurologist, who noted the left abducens palsy and an area of paresthesia and anesthesia of the right side of the lips, chin and right cheek. Dr. Ruhberg suggested the possibility of a nasopharyngeal tumor.

Dr. Schons was requested to make X-ray plates of the skull, and he reported as follows: "Lateral stereoscopic films of the head show a definite destructive process involving the posterior portion of the floor and the dorsum of the sella. The appearance is that a definite growth has eroded through and is involving the posterior portion of the sphenoid sinus, the anterior portion of the sphenoid sinus being uninvolved and showing a sharp demarcation from the mass in the posterior portion. The upper portion of the dorsum sellae is not completely destroyed but the posterior clinoid processes appear to have been destroyed. The sella is not increased in depth or in the anteroposterior diameter."

At this point the patient requested consultation at Rochester. They reported a nasopharyngeal epithelioma, Grade 4, with involvement of the sphenoid and sella. At the present time he is receiving radium treatment at their hands.

DR. V. J. SCHWARTZ reported a group of laryngeal cases, including one of Vincent's angina of the epiglottis, one of a tuberculous lesion of the vocal cord, and several cases of carcinoma of the larynx.

DISCUSSION.

DR. H. O. COOPERMAN said he wished to add a word about these laryngeal cases mentioned by Dr. Schwartz. He had a patient, a man who became progressively hoarse. When seen, his right vocal cord was in cadaveric position. He was thoroughly examined and nothing was found either locally or in the chest to account for the condition. The patient went to Rochester, and Dr. Cooperman had a report from Dr. Figi that he could find nothing more. No treatment was instituted and function gradually returned. At present the patient's vocal cord is normal. Dr. Cooperman said these cases are rather rare, and may rather frighten one a little if he cannot find anything that is pressing on the recurrent laryngeal nerve.

DR. DOUGLAS WOOD asked Dr. Cooperman which cord of the larynx was involved.

DR. COOPERMAN said it was on the right.

DR. WOOD said he had seen a case of paresis of the right vocal cord following typhoid, and that case recovered.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Meeting of May 16, 1932.

DR. HERSCHEL EZELL reported a case of angioma cavernosum, Miss L. O., age 25 years, who complained of a painful enlargement over the right cheek, which had been present since she was three years of age. The mass had been much enlarged at times and at intervals it was so small as to be hardly noticeable.

Dr. Ezell's examination revealed a tumor one-half the size of a golf ball occupying the space between the nose and eye of the right cheek, extending outward to the margin of the malar process and downward to within a fourth of an inch of the right corner of the mouth. The tumor was pale blue with a purple inflamed area slightly below its center projecting outward somewhat. The mass was soft, movable and painful to touch. The right lacrimal apparatus was normal. The nose and throat were normal. On aspiration 2 c.c. of bloody fluid was withdrawn from the tumor.

Dr. Ezell, upon communication with Dr. W. B. Anderson, who was present at the birth of the patient, learned that the tumor had been observed at the time of birth and that the opinion of several consultants had been that the growth was angioma.

Meeting of June 20, 1932.

Removal of a Deciduous Tooth from Left Anterior Ethmoid Region in Patient Forty-one Years of Age. Dr. A. N. Hollabough.

Dr. Hollabaugh reported the removal of a deciduous tooth from the left anterior ethmoid region of a patient, Mrs. D. W., age 41 years. The patient complained of headaches, especially in the top of her head, which had occurred all of her life.

Dr. Hollabough's examination disclosed profuse polypi in the left nostril and complete absence of light by transillumination of the left maxillary sinus. The other sinuses were clear.

Under local anesthesia Dr. Hollabough went about removing the nasal polypi and finding an attachment in the left ethmoid region was biting in to the left ethmoid cells when the tooth was dislodged and passed in to the patient's throat. Patient spit the tooth out.

Dentist who examined the tooth stated that it was a deciduous upper molar tooth.

Misplaced Third Molar. Dr. J. L. Bryan.

Dr. J. L. Bryan reported a case of a misplaced third molar. The patient, B. J., age 21 years, had complained of the left ear being stopped up for six weeks prior to his visit to Dr. Bryan. The condition had developed shortly after he had had an acute cold. There was slight tinnitus; there had been no pain, or headache and there was no nasal, or aural discharge. There had been no similar attacks previous to this one.

The examination revealed an inflamed nasal mucus membrane with hypertrophied inferior turbinate; the septum was deviated to the left, blocking the passage. Transillumination showed the maxillary sinuses cloudy. The tonsils were moderately septic. The ears were negative, except for a dull and retracted membrana tympani in the right and inflamed and bulging in the left.

A dental examination disclosed a fistulous tract behind the left upper second molar through which pus exuded. X-ray showed a foreign body, probably a tooth in the left antrum. The left upper second molar was extracted and a Caldwell-Luc antrotomy was performed. A tooth was found growing from the external wall of the left maxillary sinus about midway between the anterior and posterior walls, 1 inch above the floor. A mastoid gouge was used to remove the tooth. The antrum was lined with a thickened fibrous membrane. The third molar on the upper left side had not erupted, however; the X-ray showed no unerupted tooth in this position. It was, therefore, concluded that the misplaced tooth was the third molar.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of March 16, 1932.

Discussion of Symposium on Otolaryngology.

DR. SAMUEL J. KOPETZKY: In opening the discussion on a program of such varied topics, it is not my purpose to review or stress what each speaker has brought out. An otolaryngological department in a general hospital provides opportunities for special studies which take the specialist somewhat afield from the specific organ to which he is devoting himself and give him a rounded medical experience, basing his judgment and conclusions on certain broad cultural studies which develop his powers in diagnosis and prognosis. Some of the studies presented at the hospital this afternoon and described here tonight form a valuable contribution to our working knowledge. One cannot build scientifically unless legend and tradition are analyzed and what is worthless discarded. It is very helpful to have the laboratory check our clinical data; everything comes under review in the laboratory.

The studies presented by Dr. Thaler this evening are of great interest. It has been proven that hypercholesteremia is reversible in animals; in human beings it is not, and a definite degree of deafness has been found associated with the condition. In most instances the patient is unaware of the deafness, and reduction of the cholesteremia does not influence the hearing loss.

The studies of blood cholesterol in ozena and atrophic rhinitis were undertaken to substantiate or disprove the relationship between cholesteremia and the nasal conditions named. It is refreshing to know that we can with impunity discard the whole story of cholesteremia in ozena and atrophic rhinitis.

In discussing the blood picture in the treatment of septicemia, I shall stress but two points: the necessity for studying the entire picture, and the necessity for making serial studies. I still come across the occasional, casual blood examination. That is as useless as taking the temperature on Sunday and again on Saturday night of the following week. If blood studies are to be taken seriously, they must be made regularly. Study of the staff or band cells in relation to sedimentation provides valuable data. As has already been said, these last two tests are not specific; but they furnish a warning to those who use them when things are not going well with the patient and put the doctor on guard.

As to biopsies, I believe that the common habit of accepting a laboratory report from even a most distinguished tissue pathologist is not entirely in the interest of the patient if procedures are adopted based on that one reading only. I believe that the best interests of the patient are served when sufficient material is removed at biopsy for the findings of the pathologist to be corroborated and checked. We need more accuracy in handling intrinsic tumors of the larynx. As our technique develops we shall be called upon to decide whether a given case shall be submitted to one or another form of treatment—whether it shall be treated surgically or submitted to radium or X-ray therapy; and tissue pathology is going to play a larger role in the differentiation of cases to determine what therapeutic agent shall be employed. Eventually we shall expect a report from the tissue pathologist on the presence or absence of radio-active cellular elements in the tissues. This problem has not yet been satisfactorily solved.

I do not know of any contribution to otology of more value than the study of the subcultures of the invading bacterial flora in otitic infections; and I do not know of any studies that are receiving less attention. I realize that such studies in subcultures have been tried and discarded as of no practical value in some laboratories. I do not agree with this decision. When one becomes accustomed to correlating the subculture findings with the clinical course of the case, much help is derived from the knowledge of the exact "subculture" type of the invading organism because there seems to be a distinct relationship between certain subcultures and the clinical course of the

disease. There also seems to be a definite relationship between certain subcultures and the mortality rate, irrespective of the adequacy of the surgery employed. I am assuming that the surgery in every case is adequate as one must have a common starting point. In other words, in my own hands, doing the same operation in cases with all other things equal but with different types of subcultures, different results are obtained. I feel at this time that study of the subcultures furnishes practical information which can be utilized at the bedside because the data comes to you at a point in the clinical course of the case when decisions are still to be made. There is much research work done that is theoretically correct and interesting in itself but that has no practical value. I have found this particular work of great practical value at the bedside. These observations are not the result of hastily accumulated data but are the product of over six years' experience with these subcultures.

I wish to express to the officers of the Section my thanks for the privilege of presenting the work of my associates and myself this evening.

DR. JOHN A. KILLIAN: The presentation of several biochemical papers on this evening's programme affords to the biochemist great satisfaction. The indicated collaboration between clinicians and the biochemist is exactly as it should be. It is particularly encouraging to witness the wealth of detailed knowledge of biochemistry displayed by clinicians in this comparatively narrow field of medical science.

While listening to Dr. Thaler's presentation, a number of questions suggested themselves to me. As an analyst, I am interested in the methods employed in the determination of the cholesterol of the blood because in the evaluation of the significance of changes in cholesterol in blood, the factor meriting primary consideration is the analytical method employed. If the Myers-Wardell procedure was followed, the figures reported by Dr. Thaler are definitely above normal. On the other hand, the results are merely high normals for the Bloor method. Moreover, Somach and Halpern have shown significant variations in the cholesterol of the blood for normal subjects and some hospital patients when analyses were made at intervals of one hour and for a twenty-four-hour period. It appeared to me that the figures presented by Dr. Thaler did not vary from the accepted normal range more than the diurnal deviations of cholesterol from the mean for the twenty-four-hour period. Furthermore, the cholesterol of the blood alone is never changed. Within our experience, changes in blood cholesterol are accompanied always by other changes in the blood. It is not evident that Dr. Thaler has excluded these other blood changes as potential factors affecting auditory acuity. If hypercholesterolemia diminishes auditory acuity, it is logical to expect hypocholesterolemia might increase auditory acuity. Hence, Dr. Thaler's results might have been made more conclusive by a comparison of auditory acuity in subjects with hypercholesterolemia with the acuity in cases of hypocholesterolemia.

With Dr. Wachsberger and Dr. Fishberg, I believe that Fleischmann's reports of a markedly decreased blood cholesterol in atrophic rhinitis are incredible. During fifteen years' experience in blood analyses, we have not observed in any pathologic state a fall in blood cholesterol to anywhere near the level of 20 mg. per 100 ml. In a small group of cases of atrophic rhinitis, our figures for blood cholesterol have been between high and low normals by the Sackett method.

Dr. Fishberg's essay was a comprehensive resume of contributions by biochemists to otology. The general conclusion to be drawn from this critical summary is that to date these investigations have yielded no results of any value from a practical standpoint. Some years ago our laboratory made an intensive study of the blood changes in otosclerosis. The research Fellow making the study was himself a subject of otosclerosis. All the blood constituents studied were within normal limits. The laboratory worker was so discouraged that he refused to publish his results. This is a lamentable attitude to adopt towards analytical results in any investigation. It is illogical to expect that a slow process would produce changes in the blood demonstrable with our present methods of analysis.

I would like to emphasize the points expressed by Dr. Fishberg . . . future investigations beginning where these have left off, guided by past experience

and using more refined methods will produce probably results of more practical value from etiologic and therapeutic standpoints.

Dr. Andrew A. Eggston: We have had some experience with different phases of the subject taken up in the programme this evening, at the Manhattan Eye and Ear Hospital in the work Dr. Fowler has carried out in chronic infections of the ear. We have examined many cases, biochemically and clinically. The laboratory examinations consisted of blood sugar, cholesterol, calcium and phosphorous. In none of the cases were the findings of significance. There may be deafness when there is hypocholesteremia, but not necessarily hypercholesteremia with deafness. The calcium and phosphorous show no significant changes. From a biochemical study we found only negative results in the question of deafness and otosclerosis.

As to the atrophic rhinitis, we have done no chemical studies, but our studies of the mucous membrane has led us to the opinion that the pathological processes involved have not received sufficient study. We feel that atrophic changes in the mucous membrane are secondary to the vascular changes, particularly arteritis. Blood counts in otology are not of as much significance as in general surgery. The mastoid diseases do not give the changes, as, for instance, in appendicitis. In regard to the stab cells, or immature polys, as we like to designate them, with children the immature cell count is of some value in revealing a septic factor. It probably also indicates the extent of the sickness of the patient, whether seriously ill or only slightly so. This is also true of the sedimentation test. Neither have any specific value; both should be done serially. Both tests represent a refinement in examination and should have some practical value.

I am grateful to Dr. Plaut for his discussion on the biopsies of the larynx. There should be a closer co-operation with the internists in the examination of pathological material. Consultation between the pathologist and the internist will certainly aid in the correct diagnosis and interpretation of lesions. There is no field where this is more important than in the larynx, where such small pieces are examined and where the consequences are so grave. I agree with Dr. Plaut that many of the polypoid cases have been erroneously diagnosed angiomata. Nearly all the polyps are edematous tissue and are usually the result of venous obstruction, such as occurs in hemorrhoids. I noticed that Dr. Plaut did not refer to Broder's classification of malignancy. We do not use Broder's classification, but always give an opinion as to the degree of malignancy as suggested by the infiltration and inflammatory reaction, nuclear changes, etc., which has always been done before Broder's work. The point that Dr. Plaut makes, that the pathologist can help in determining the ray sensitiveness of tumors is very important and therefore should be consulted in the type of treatment to be employed.

As to the value of typing subcultures of streptococci in otology, I recall that in 1920 we began typing all the cultures from the ears according to Holman's classification, and for five years we continued this study, but as nothing of practical value was obtained, the work was discontinued. There are many different methods of typing streptococci and, accordingly, so many different types of streptococci, that the subject is very difficult. As Holman's classification depends primarily upon whether the organism in question is hemolytic or nonhemolytic and as this faculty on the part of streptococci is quite variable, it renders the results of limited value. The prognostic value of the types of streptococci is also important as the question of the resistance of the patient must enter into consideration of the course of an infection. Further in our studies we found that the type of organism varied from the earlier to later stages of the infection and many of the bacteria early are contaminating bacteria. It is nice work and very interesting, but from a practical standpoint it is rather limited in value.

Dr. Marshall C. Pease: There is need for caution here tonight. The clinician feels somewhat lost in this group of specialists and especially in discussing a series of highly technical papers by a group of laboratory workers. Perhaps a very practical clinical person is needed in such a discussion in order to keep the meeting on a practical basis. In the first place, I would congratulate the Section on the papers as presented. I have sat here for two and one-half hours fascinated by these presentations and with not the slightest inclina-

tion to go to sleep, but it must be confessed that from the standpoint of the patient I have received not the least help. There has been nothing said here tonight that will be of any, or at least that can be of more than little help to me as I stand at the bedside, either in diagnosis or in treatment.

The work in blood chemistry, so far as I know children, has not helped even remotely. I do not deprecate this work. I think it should be done and I agree with Dr. Kopetzky that even negative work should be published, if for no other reason than to make it unnecessary to repeat such works.

It is some question as to whether the study of immature cells or as to whether the sedimentation test will be useful to me as I look down the narrow canal of a child's ear trying to decide whether or not it has an otitis media and if it has to answer the further question of whether or not it should be immediately opened. I doubt whether such test will greatly assist in that further and more important matter of deciding whether or not the mastoid should be opened. These are important matters and we need help in properly answering these questions.

Perhaps the subcultures of the infecting organisms may be of help. However, I may say that I have done some culturing of the streptococcus group in my day, so that I have a clear notion of how mixed these organisms are and of how large a group of bacteria are normally found in the ear. There is one feature of this work to which we might profitably pay more attention and that is in the taking of cultures when the drum is opened—I believe we are missing some valuable information at this point in our procedure.

In the matter of biopsy material the most important fact brought out was that, after all, the clinician has to decide the matter. I have done enough laboratory work to know that the fact that you do not find a certain pathological condition does not mean that it does not exist. It simply means that you have not found it. A negative finding never has the value of a positive finding. I had hoped that the importance of autopsy material in this field might be emphasized. There is so much that we don't know about the nose and ears of children and to which we badly need an answer that the collection of such material is of the greatest importance. Every effort should be made to have the ears and sinuses carefully examined in every child that comes to autopsy. This is true not only of the unoperated case but even perhaps more true of those which have been operated upon. The entire subject needs to be re-examined without prejudice and without being too greatly hindered by our preconceived notions.

Perhaps the one great thing that comes out of this meeting tonight is the desirability of more frequent consultations at the bedside of the surgeon, pathologist and clinician. They can be of mutual benefit to each other. We will know more about the case and we will know better how to treat the next one. There is perhaps no place in medicine where there is a greater need for careful and the exchange of ideas than in the infections in children around the nose and ears.

Do not misunderstand me in this discussion. It has been a good and interesting evening and it is well within the truth to say that on the whole it has been a profitable evening. It is proper, however, to add that there has been nothing present here which will be of an assistance either in diagnosis or the treatment of upper respiratory infections in children. This maybe appears to be a very utilitarian point of view but the point is made deliberately in the full recognition of the present inadequacy of the diagnostic methods and treatment of infections around the nose and ears of children. There is no place in all medicine today insofar as children are concerned where the diagnosis is so regularly inadequate and the treatment so commonly inefficient as in infections of the sinuses and mastoids in children, and especially in babies.

DR. NATHAN THALER: Replying to Dr. Killian, I would say that we did not investigate hypocholesteremia cases. In the second place, blood determinations were not done with sufficient frequency to determine diurnal variations; but in those cases where the examinations were repeated they were uniformly high. We indiscriminately did hearing tests on hypercholesteremia cases to check the pathological findings and found the association as reported.

DR. ELLA H. FISHBERG: Replying to Dr. Thaler, the demonstrations were done according to the Autenrieth-Funk method.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Regular Meeting, May 18, 1932.

Presentation of New Instrument: Scissors-Hemostat. Dr. A. F. Warren.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. ROBERT H. FOWLER: May I say a word in favor of this instrument introduced by Dr. Warren? It seems to be the exception that proves the rule. I have always been wary of combining two functions in one instrument, like putting an automatic pair of legs on a golf bag to take the place of a caddy, but in this case the two functions of this one instrument—grasping forceps and cutting the catgut tie—seem to be happily combined so that neither interferes with the other.

Radium in Malignant Tumors of the Nasal Sinuses. Dr. G. Allen Robinson.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. JOHN MCCOY: What was the amount of radiation given to these patients?

DR. G. A. ROBINSON: In regard to the dosage used in the treatment of malignant tumors of the nasal sinuses, the earlier cases were treated more or less empirically. At the present time a more accurate dosage is employed. If the bony walls are not involved the tumor mass may be considered a sphere 3 c.m. in diameter; if the walls are involved, a sphere of 4 c.m. According to a dosage table sufficient radium is employed to deliver six to ten erythema doses to the tumor. The total number of milligram hours varies from 3500 to 6000.

Correction of Post-Auricular Defect by Implantation of Fascia Lata. Dr. Charles M. Griffith and Dr. Albert Schattner.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. GLUSHAK: The implantation of fat has interested me very much and I have had some personal experience with the procedure. I have looked up several authorities on the subject and find their opinion as to the durability of the fat implanted vary considerably. The majority are inclined to believe that the fat is absorbed, either entirely or to a great extent, so that the quantity implanted should be at least twice the amount necessary to fill the depression. Lately it has been advisable to procure fat attached to fascia which is supposed to prevent much absorption of the fat. In my experience with one case of a frontal sinus depression where I inserted an exaggerated piece of fat attached to fascia lata, the fat was absorbed to the extent of about three-quarters of its original quantity and resulted in a smaller depression than at first. The resulting depression was still ugly.

I also had occasion to close a depression in a previously operated mastoid and this time used cartilage from patient's own ear. This result was very gratifying. In these mastoid depressions, if the bottom is bony a solid substance like cartilage or ivory may be used with success.

I was much interested in this case of Dr. Griffith's and unfortunately he failed to give a proper description of the depression as to its depth, etc., and the technique of implantation of the fat.

DR. C. M. GRIFFITH: I cannot give the exact depth of the depression, but it depends on how deep the mastoid was. How much shrinkage there will be, I don't know. The case has been healed for a couple of months, but whether it will shrink more I cannot say. Dr. Imperatori has asked me to present the patient again in the fall.

A Case of Laryngectomy Fourteen Years Ago. Dr. John McCoy.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Nasopharyngeal Malignancy Case Report. Dr. E. H. Moyle.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Otitic Infection with Gastroenteritis in Infants. Dr. Max Rabbiner.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Stylohyoid Ossification Bilateral and Injury of Ninth Nerve After Tonsillectomy. Dr. Robt. H. Fowler.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. IRVING PARDEE: The neurological examination of this patient shows the following: There is a lack of tone in the muscle of the soft palate above and around the tonsil. These muscles in addition to their hypotonia lag on attempted movement, the palate being drawn to the right. The gag reflex is diminished on the left side of the pharynx to all stimuli. The sensory examination of the throat reveals a diminished sensation to touch and to pain, i. e., pin prick, on the soft palate, tonsils, and the back of the palate—though there is a spot of hyperesthesia on the anterior portion of the left tonsil. There is no sensory disturbance of this nature on the tongue. Taste was examined with some difficulty but the following observations allow us to draw some conclusions: Subjectively he complains of a salty taste which goes over his entire mouth and he notes that he is unable to taste the difference between a good and a poor brand of tobacco. The taste for salt is not appreciated on the posterior half of the left side of the tongue, while sweet is diminished, although appreciated. The taste for cinnamon is preserved on the posterior third of the tongue but its recognition is distinctly diminished as compared with the opposite side of the tongue or the anterior portion.

Under these circumstances I feel that we have here the first opportunity to study the functions of the glossopharyngeal nerve, an opportunity which has not presented itself insofar as I know in the medical literature; most of the studies having been made on intercranial lesions involving the ninth nerve but not on peripheral lesions.

Owing to a very short opportunity to study this case before this meeting, I feel that I may have to make some revision of this sensory examination as it will have to be repeated a number of times before I am convinced especially of the disturbance of taste. However, I believe that we have definite confirmatory evidence here that this patient is suffering from an isolated incomplete paralysis of the glossopharyngeal nerve and it proves conclusively that this nerve must supply motor fibres to the muscles elevating the soft palate and also that its sensory representation involves the soft palate, tonsils and back of the pharynx in a larger area than has hitherto been considered its distribution. Dr. Fowler has made an unique contribution in demonstration of the probability of injury to the nerve in a tonsillectomy and his anatomical presentation I would not presume to discuss, as he has studied it in so great detail. The neuroanatomical findings in this case are exceedingly interesting to me and will represent as valuable a contribution to the physiological function of this nerve as Dr. Fowler's observations have to you, before whom lies the possibility of injury to this nerve.

DR. LORE: Since Dr. Fowler mentioned to me the ninth nerve as being in close relation to the lower pole of the tonsil I have had a chance to look this up on dissection and have found that by exposing the ninth nerve near where it enters the base of the tongue it was approximately on the capsule. To test this out I placed the first finger of one hand inside the tonsil fossa and the first finger of my other hand against the ninth nerve from outside, rubbing the nerve between the two fingers. The point at which it is apt to be injured seems to be where we go back to remove the lingual prolongation, the bit of tonsil tissue at the base of the tongue, so my investigation bears out what has been said.

The point that Dr. Fowler raised in reference to the ninth nerve merits some serious consideration. There is no doubt in my mind that at times this

nerve may be injured when the so-called lingual tab is removed. Not infrequently on the removal of this tab of lymphoid tissue one may go considerably deeper than is necessary and it is not inconceivable that the nerve may be either injured or drawn into the wound by subsequent scar tissue formation.

DR. FOWLER (closing): If one were anxious to demonstrate the proximity of the ninth nerve to the lower lobe of the tonsil and its vulnerability during procedures such as electrocoagulation or the surgical removal of tonsils, what could be more convincing than to have the presence of a stylohyoid ossification furnish a rigid framework encompassing the field? If one desired to differentiate between injury to this nerve from that of any other nearby nerve such as the lingual, what better arrangement than to have a styloid process reaching all the way to the hyoid? And if Dr. Pardee is right in stating that the symptoms in this case imply an isolated incomplete paralysis of the glossopharyngeal nerve, it is fair to imply that, more than we have ever been given to understand, the ninth nerve is vulnerable.

The Use of Avertin in Endoscopy. Dr. Geo. R. Brighton.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. WILLIAM BRANOWER: I have used avertin, mostly in operations for thyroidectomy. The most important point I wish to bring out is that it is used as a basal anesthetic only, and if used in the proper dosage there is no danger of respiratory paralysis. I have gone over 300 cases in which avertin was used and found that too large doses had been used. Eighty milligrams, supplemented by nitrous oxide and oxygen, will give no trouble.

DR. GEORGE R. BRIGHTON: I am glad Dr. Branower brought up the point he did, for we found that as a basal anesthetic it works beautifully in esophagoscopy without the addition of a supplementary anesthetic. In some of the cases we used a few drops of ether, but in later cases we have given smaller doses with very satisfactory anesthesia.

Tonsillectomy in the Presence of Thyroid Disease. Dr. H. P. Bullwinkel.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HERMAN JARECKY: I had the pleasure recently at the Harlem Medical Association of listening to Dr. Crile on toxic thyroids. Both he and Dr. Henry Louria, who is in charge of the thyroid clinic at the Brooklyn Jewish Hospital, in the discussion, advised in such conditions that tonsillectomy should follow a month after the thyroidectomy.

DR. BULLWINKEL (closing): There are two factors to consider: First, the upset of the polyglandular balance; second, the opening of the vascular and lymphatic channels to infection.

Some Clinical Observations in Labyrinthitis. Dr. Daniel S. Cunning.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. MAYBAUM: Dr. Cunning has stated in a precise and accurate manner the problem concerned in surgery of the labyrinth. It is difficult to decide when to operate on the labyrinth. We are not always certain at the start whether we are dealing with a purulent or a serous labyrinthitis. Only by careful observation of the patient, noting an increase or diminution of symptoms, can we determine the difference. To operate too early or too late may result in a purulent meningitis. The diagnosis of a suppurative labyrinthitis does not immediately mean that an operation is necessary. Quite the contrary. American otologists have taken issue with the European assertion that such cases should be interfered with early. We feel that it is far better to wait until the suppurative process is localized, rather than operate early and cause a spreading meningitis.

At the beginning we are dealing not only with a suppuration of the labyrinth but with a localized serous labyrinthitis. By carefully observing the patient as to the early manifestations of meningitis, together with repeated lumbar puncture findings as a guide, indication for intervention can be reached.

Rarely suppurative labyrinthitis may heal spontaneously. Ten or twelve years ago I had occasion to be present at an operation for a supposed suppurative labyrinthitis. A radical mastoidectomy had been done. A search for a semicircular canals revealed the fact that these were not present; in other words, the labyrinth had healed spontaneously. A dead labyrinth in itself is not an indication for a labyrinthectomy. This patient had had severe labyrinthine symptoms over two years previously and from then until the time of operation did not have manifestations of labyrinthine irritation. Obviously there was no reason for a labyrinthine operation in this case.

DR. M. D. LEDERMAN: I do not think Dr. Cunning wishes the impression to go out that a fulminating labyrinth is a common condition. We have all seen many cases of chronic suppurative middle ear disease, but comparatively few cases of fulminating labyrinthitis occur. I have had my share of chronic suppurative cases and think I can count on one hand the number of cases of fulminating disease that have occurred in the course of a suppurative otitis media while under observation. No less an authority than Dr. Joseph Beck states very positively that he has never seen a case of serious involvement of the labyrinth while the patient was under his care for suppurative otitis. I believe a conservative attitude is the one to be observed. Some of the cases reported in my paper on the "Non-Surgical Dry Treatment of Chronic Suppurative Otitis with Iodine Powder (Sulzberger), THE LARYNGOSCOPE, June, 1930, had symptoms of vestibular irritation, and as the suppuration improved these annoying symptoms disappeared. One of the patients, a young woman, had had an infection of the middle ear for five years. For two months previous to my examination she complained of dizziness daily, off and on. This symptom was much increased on douching the ear, and became so serious that she was afraid to go out alone. Attention to the pathology in the middle ear and regular treatment with the iodine powder resulted in the ear becoming dry and the disappearance of the vertigo. At the onset the prognosis was very dubious, but regular treatment with the powder and avoidance of irrigation brought about a very happy result with useful hearing function.

DR. HUGH BLACKWELL: The point to be stressed in connection with this subject is that labyrinthine irritation presents the same symptoms as acute labyrinthine invasion in its early stages. Cases presenting labyrinthine symptoms should have performed, when necessary, either an immediate radical or a simple mastoid operation. These procedures will frequently cause the symptoms to disappear by removing the source of irritation. For a number of years I have followed this practice, and during this time have not found it necessary to perform a single labyrinthine operation. In addition, in those patients who have evidence of cortical labyrinthine invasion, as shown by the presence of fistula or complete loss of hearing, I have in two instances observed the aural function return following these operations, without having performed a labyrinthine operation.

(To be continued.)